

THE BRITISH
JOURNAL OF SURGERY

THE BRITISH JOURNAL OF SURGERY

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EPONYMS

BY SIR D'ARCY POWER, KBE, LONDON

V. SIR JAMES PAGET.

THE name of Sir James Paget is associated with a disease of the nipple and with a remarkable change in the skeleton to which he gave the name of 'osteitis deformans'. It is noteworthy that the true pathology of neither of these conditions has yet been worked out.

ON DISEASE OF THE MAMMARY AREOLA PRECEDING CANCER OF THE MAMMARY GLAND

The paper on "Paget's Disease of the Nipple", as it is now commonly called, appears in the tenth volume of *The St Bartholomew's Hospital Reports* for the year 1874, pages 87-9. As a classical contribution to surgery it is even shorter than Abraham Colles's description of the fracture of the wrist with which his name is now associated.

Paget writes: "I believe it has not yet been published that certain chronic affections of the skin of the nipple and areola are very often succeeded by the formation of scirrhus cancer in the mammary gland. I have seen about fifteen cases in which this has happened, and the events were in all of them so similar that one description may suffice.

'The patients were all women, various in age from 40 to 60 or more years, having in common nothing remarkable but their disease. In all of them the disease began as an eruption on the nipple and areola. In the majority it had the appearance of a florid, intensely red, raw surface, very finely granular, as if nearly the whole thickness of the epidermis were removed, like the surface of a very acute diffuse eczema, or like that of an acute balanitis. From such a surface, on the whole or greater part of the nipple and areola, there was always copious, clear, yellowish, viscid exudation. The sensations were commonly tingling, itching, and burning, but the malady was never attended by disturbance of the general health. I have not seen this form of eruption extend beyond the areola, and only once have seen it pass into a deeper ulceration of the skin after the manner of a rodent ulcer.

'In some of the cases the eruption has presented the characters of an ordinary chronic eczema, with minute vesications, succeeded by soft, moist, yellowish scabs or scales and constant viscid exudation. In some it has been like psoriasis, dry, with a few white scales slowly desquamating, and in both these forms, especially in the psoriasis, I have seen the eruption spreading far beyond the areola in widening circles, or, with scattered blotches of redness covering nearly the whole breast.

I am not aware that in any of the cases which I have seen the eruption was different from what may be described as long-persistent eczema, or psoriasis, or by some

other name, in treatises on diseases of the skin and I believe that such cases sometimes occur on the breast, and after many months duration are cured, or pass by and are not followed by any other disease. But it has happened that in every case which I have been able to watch, cancer of the mammary gland has followed within at the most two years, and usually within one year. The eruption has resisted all the treatment, both local and general, that has been used, and has continued even after the affected part of the skin has been involved in the cancerous disease.

"The formation of cancer has not in any case taken place first in the diseased part of the skin. It has always been in the substance of the mammary gland, beneath or not far from the diseased skin, and always with a clear interval of apparently healthy tissue.

"In the cancers themselves I have seen in these cases nothing peculiar. They have been various in form—some acute some chronic—the majority following an average course, and all tending to the same end, recurring if removed, affecting lymph glands and distant parts showing nothing which might not be written in the ordinary history of cancer of the breast.

"The single noteworthy fact found in all these cases is that which I have stated in the first sentence, and I think it deserves careful study. For the sequence of cancer after the chronic skin disease is so frequent that it may be suspected of being a consequence and must be always feared, and may be sometimes almost certainly foretold. I believe that a nearly similar sequence of events may be observed in other parts. I have seen a persistent 'rawness' of the glans penis, like a long-enduring balanitis followed after more than a year's duration by cancer of the substance of the glans. A chronic soreness or irritation (of whatever kind) on the surface of the lower lip often long precedes cancer in its substance, and with a frequency surpassing all other cases of the kind the superficial syphilitic discolorations of the tongue are followed, and not superseded by cancers which do not always appear to commence in a diseased part of the tongue.

"For an explanation of these cases it may be suggested that a superficial disease induces in the structures beneath it, in the course of many months, such degeneracy as makes them apt to become the seats of cancer, and that this is chiefly likely to be observed in the cases of those structures which appear to be naturally, most liable to cancer—as the mammary gland the tongue, and the lower lip. One may suspect that similar surface-irritation has much to do with the frequency of cancer of the rectum, pylorus, and ileo-cæcal valve in any of which parts the degeneracy, which might come naturally in old age and make them apt for cancer, may be hastened, and made prematurely sufficient, by an adjacent disturbance of nutrition.

"In practice, the question must be sometimes raised whether a part through whose disease or degeneracy cancer is very likely to be induced should not be removed. In the member of a family in which cancer has frequently occurred and who is at or beyond middle age, the risk is certainly very great that such an eruption on the areola as I have described will be followed within a year or two by cancer of the breast. Should not then, the whole diseased portion of the skin be destroyed or removed as soon as it appears incurable by milder means? I have had this done in two cases but I think too late. Or, again, when one with a marked family-habit to cancer has syphilitic disease of the mucous membrane of the tongue, with frequent recurrences of inflammation—should not all the worst pieces of the membrane be removed? I should certainly advise it especially if the membrane were ichthyotic, if it were not that the disease is commonly so extensive that good scar-tissue would not be likely to be formed and that bad scar-tissue often irritable and ulcerating, is as likely to induce cancer as the syphilitic or ichthyotic patches would have been.

The publication of this paper proved a matter of interest both clinically and pathologically. Those who saw the actual cases and followed up the subsequent course of similar ones were clear that this form of chronic inflammation did not always end in cancer, and that, as Paget stated local excision was sometimes followed by cure. It was

Lecum of the nipple in the right breast, occurring three years after removal of the left breast for scirrhus

From a drawing by Thomas Goddard March, 1881,
in the Museum of St Bartholomew's Hospital

recognized, however, that cancer occurred very frequently, and the condition was looked upon as 'precancerous'

The present view held by the majority of surgeons is that cancer of the breast precedes the eczema of the nipple and causes it. Mr Sampson Handley presents this explanation in the following words (*The British Journal of Surgery*, 1919-20, vii, 189) "A carcinoma starts in the smaller ducts of the breast, perhaps exceptionally from the acini or the larger ducts. Usually, without producing a palpable tumour, it permeates the breast lymphatics widely. The rich plexus of lymphatic vessels around the ducts forms an especially easy and convenient channel for permeation, and the lymphatic block extends along them to the subareolar plexus beneath the nipple. The cutaneous lymphatics about the nipple are now dammed up so that lymph cannot return from them. Later they are themselves permeated, but possibly this is not always the case. At this stage and before any lump has appeared in the breast, the skin of the nipple and the mucosa of the ducts begin to show changes dependent upon lymphatic obstruction. The epithelium shows disintegration and degeneration of its superficial layers with proliferation of the deeper layers. These changes are nutritional and non-malignant. The dermis becomes thickened by solid lymphatic oedema. In the rare cases where no carcinoma has made its appearance though the Paget's disease has lasted many years, it is probable that an atrophic scirrhous which may have undergone partial or complete cure, preceded the onset of the Paget's disease. But the possibility that the lymphatic obstruction in such cases is of inflammatory origin and due to a chronic lymphangitis cannot be altogether excluded.

It will be noticed that Paget's original paper dealt entirely with the clinical aspects of the disease. In 1875—a year after the publication of the memoir—the histological details of two similar cases were described in *The Medico-Chirurgical Transactions*, lix, 107, by Butlin who added two more in the course of the following year.

Matters rested there for some years and as the disease is rare little notice was taken of it until on June 4 1890, Louis Wickham read as his thesis for the Doctorate of Medicine at Paris a 'Contribution a l'Etude des Psorospermoses cutanees et de certaines Formes de Cancer Maladie de la Peau dite Maladie de Paget'. The thesis opened with the bold statement 'La maladie de Paget est une affection parasitaire du groupe des psorospermoses cutanees, caracterisee par l'inflammation chronique de la peau, des glandes et de leurs conduits, suivie de proliferation epitheliale.' ("Paget's disease is parasitic, the result of cutaneous psorosperms, and characterized by a chronic inflammation of the skin, glands, and ducts associated with epithelial proliferation.") The thesis which was clearly inspired by Darier who was the head of the Laboratory at the Hopital Saint Louis, quickly attracted attention throughout Europe and America, and the battle of cancer parasites raged round Paget's disease of the breast for several years. Many observers claimed to have discovered the true parasite of cancer, but no two agreed upon the same, and after a few years the controversy died away. It proved of lasting value, however, because it led skilled histologists to investigate the changes—degenerative and otherwise—which take place in epithelial cells and many forms of cell-inclusion, vacuolation and oedematous change became familiar.

The plate of Paget's disease of the Nipple is made by the kind permission of the Treasurer and Governors of St Bartholomew's Hospital, from a water-colour sketch of a patient sent to the Hospital by Sir James Paget in 1884. The drawing is No 1057 in the Museum of St Bartholomew's Hospital.

(To be continued)

REMOVAL OF INTRATHORACIC TUMOURS BY THE TRANS-STERAL ROUTE

By T. P. DUNHILL, C.M.G., LONDON

LARGE intrathoracic tumours are seldom successfully removed in their entirety, and this seems to justify the publication of the first of the cases here recorded. Two cases of intrathoracic enlargements of the thyroid gland are added, because their depth and position in the mediastinum made it necessary that they should be approached by an unusual route.

In all three instances the tumours were removed through an incision which split the upper half of the sternum. This method of approach had been practised by Professor G. E. Gask before the war. During the war, and subsequently in civil work, this operator has been perfecting methods of access to the thoracic cavity designed to give more efficient working room.¹ A trans-sternal route had also been used by Pierre Duval in order to reach the right auricle and inferior vena cava, but for this purpose he split the lower half of the sternum.² By this means he removed a bullet which kept moving to and fro in a disconcerting fashion between the patient's great vessel and heart. Lihenthal removed a mediastinal thyroid by the trans-sternal route.³

In each of the following three cases urgent necessity compelled intervention, unless the patients were to be left to their fate. The difficulty of respiration had reached a degree which, in the first case, was incompatible with exertion, and in the other two was rapidly becoming incompatible with life.

Dr J. H. Drysdale has kindly supplied the following notes of *Case 1*.

Case 1—"E. T. P., age 35, male, was sent to me by Dr von Beigen on Dec. 2, 1920, with 'symptoms of intrathoracic pressure'."

"The patient was in his usual good health till March, 1915, when he had an attack of 'influenza' with pulmonary catarrh. In Nov., 1916, and again in April and Oct., 1917, he had similar attacks. From Dec., 1918, to Feb., 1919, he had a prolonged illness with fever, occasionally reaching 102° much sputum, and violent cough. Three examinations of the sputum for T. B. were all negative. In March, 1919, he had a course of vaccines, and thereafter had been free from catarrhal symptoms, and expectoration entirely ceased. In June, 1919, however, dyspnoea—increased by exertion—became noticeable, and persisted up to the time of examination."

"Recently had suffered from 'rheumatism', chiefly in the right arm. Not losing weight."

"ON EXAMINATION—Cyanosis of the head and neck, and upper extremities. The right upper arm was 1½ in. larger in circumference than the left, and the forearm ¾ in. There was no pitting of the subcutaneous tissue nor any obviously enlarged veins. Hanging down, the right arm is distinctly bluer than the left. The skin of the right arm and hand was quite dry and almost scaly. The patient stated that he had not sweated (or hardly at all) in that arm for *twelve or fourteen years*. The grip of the right hand is feeble, but that of the left is normal. The radial pulses were equal, as were the pupils. There was no obvious deflection of the trachea nor a tracheal tug. Chest—no abnormal pulsation could be seen or felt. On the right side, from the apex down to about the 3rd rib the percussion note was considerably impaired, the impairment reaching almost to the left border of the sternum. Similar signs present over a rather larger area behind. The breath sounds were slightly stridulous on both sides, the amount of air entering being somewhat less on the right side than on the left."

"The signs pointed to the presence of a tumour occupying the mediastinum and upper part of the thorax on the right side. The symptoms, especially the prolonged absence or deficiency of sweating of the right arm, suggested some very slowly progressing lesion. Hydatid cyst or teratoma were considered to be the most likely alternatives. An operation was advised."

Shortly after being seen, and before any further examination could be carried out, the patient had another attack of pulmonary catarrh and was for some days in a condition of great danger. Wassermann's reaction, negative. Blood count, normal.

An x-ray picture was taken by Dr G Harrison Orton on Jan 12, 1921 (*Figs 1, 2*). On Jan 27 he was seen by Dr J Perkins, who agreed with the diagnosis of mediastinal tumour, and also advised operation.

OPERATION—An operation was planned which would enable an osteoplastic flap to be raised, access to the upper part of the right thoracic cavity being thus obtained. This flap was to include the right half of the sternum, from the suprasternal notch to the third intercostal space together with the clavicle and the three upper costal cartilages and ribs.

This operation was performed on Feb 19, Mr Geoffrey Keynes assisting me. An intra-tracheal anæsthetic of gas and oxygen, passed through detoxicated ether, was given with Kelly's

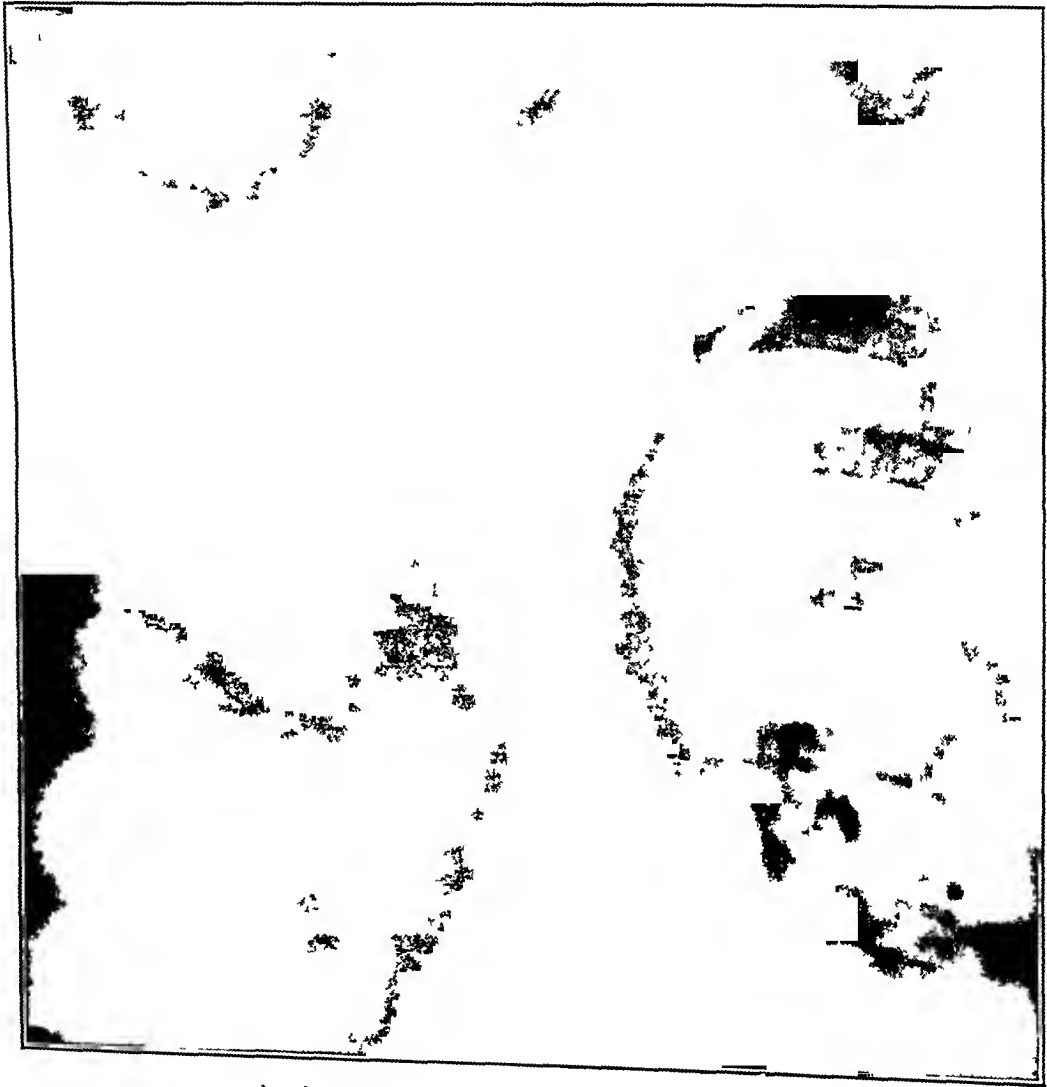


FIG. 1. Case 1. Intrathoracic fibroma. Antero-posterior view.

apparatus by Mr C.ington Hower. A skin incision was made as shown in the photograph (*Fig 3*). The upper limb of the incision was the same as that used for a goitre operation, because in the present case the opacity of the tumour had been seen in the x-ray photographs to extend higher than the right clavicle (although nothing could be felt in the neck) and it was therefore necessary first to make certain that it was not an intrathoracic goitre. The lower limit of the tumour was as low as the third interspace, but goitres sometimes descend as low as this (*Case 2, Fig 8*). The lower border of the thyroid gland was found to be unconnected with the tumour. From the centre of the incision in the neck a vertical incision was made over the middle of the sternum down to the level of the articulations of the fourth costal cartilages, and was then continued outwards to the right over the fourth rib for about five inches. The sternum was divided in the line of the

vertical incision and to the right, into the third interspace (*Fig. 4*), the intercostal muscles being also cut to the extent of the skin incision. The internal mammary vessels were ligatured and cut above and below, and the osteoplastic flap could then be used.

Access to the tumour was thus obtained. It was crossed by the junction of the right internal jugular and subclavian veins, and these had to be manipulated out of the way. The mass filled the dome of the right thoracic cavity so completely that it had displaced the pleura downwards and was fairly easily separated from this below. The tumour could then be defined, and it was found that there was barely room for a flat hand to work round between it and the ribs laterally. Behind, it was closely applied to the ribs and mesially it appeared to be firmly attached to the



FIG. 2.—*Case 1.* Intrathoracic fibroma. Lateral view to show depth in the thorax.

bodies of the vertebrae, in front of which its lower part seemed to be in contact with the base of the heart and the great vessels. Anteriorly it would have reached the chest wall but for the fact that this was lifted up and retracted away from it. Its lower convexity was overlapped by an edge of the right lung.

Since the mesial aspect of the tumour appeared to be firmly fixed, it seemed unwise to try to separate it without first obtaining the patient's permission to take the evident risk. Any tearing of the great vessels would have resulted in uncontrollable hemorrhage. The osteoplastic flap was therefore replaced and sutured in position.

A month later, March 24, permission having been given to proceed, the wound was re-opened in the same fashion as before. On this occasion Mr S. L. Higgs assisted me, and in intratracheal

anæsthetic was given by Dr. Magill. This form of anæsthesia prevented any collapse of the lung from taking place when the thorax was opened, and gave perfectly regular and comfortable breathing throughout the operation. Gradually the tumour was separated from its surroundings by working first on one side and then on another, until it appeared to be free except for an attachment to the periosteum covering the body of a vertebra. This was broken through and the tumour lifted out of the thorax. At the site of attachment there was some bleeding from a laceration of vessels; this was ligatured. When the tumour was removed an ulcer on the body of a vertebra was seen to be bare of periosteum. The induration of the pleura resulting from the traumatism of the first operation prevented the lung from expanding at once, so that a huge cavity remained. The

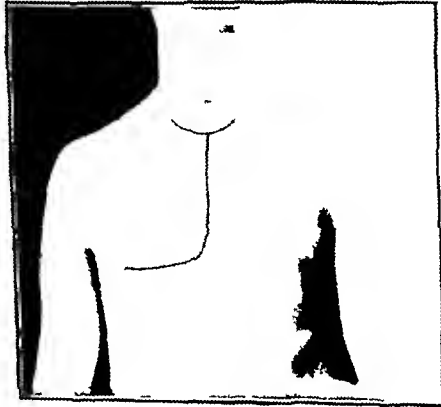


FIG. 3.—Case 1. To show the incision.

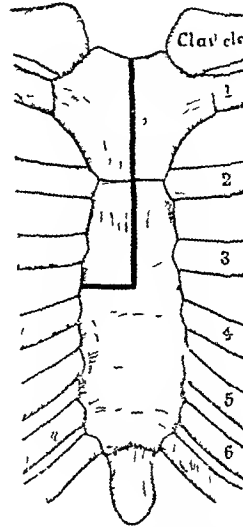


FIG. 4.—Case 1. Line of division of sternum.

osteoplastic flap was replaced and sutured accurately in position without drainage. The wound healed and, except for some itching in the arm, the patient's convalescence was uneventful. An



FIG. 5.—Case 1. Four and a half weeks after removal of tumour. Fluid in cavity.



FIG. 6.—Case 1. Nine months after removal of tumour. Fluid absorbed. Lung expanded.

physical examination by Dr. Harrison Orton on April 28, four and a half weeks after the operation, showed that the cavity was not yet obliterated, and that it contained fluid half an inch in depth.

when the patient was standing (*Fig 5*) Free movement of the fluid could be seen at its surface. A second examination on Nov. 29 showed that all the fluid had disappeared and the lung had fully expanded (*Fig 6*). The patient is now quite well, and is carrying on his business.

DESCRIPTION OF THE TUMOUR—A pathological examination of the tumour was made by Sir Frederick Andrewes and Mr Geoffrey Keynes. It was found to be a very firm, rounded, encapsulated mass (*Fig 7*). It measured about 13 cm in its greatest diameter and weighed 560 gms (1 lb 3½ oz). The capsule showed signs of having been torn away from an area of attachment about 2 cm in diameter. The tumour as a whole was relatively avascular. Microscopic sections made from several different parts of the tumour, including the area of attachment, showed that it was composed wholly of fibrous connective tissue. There were some opaque yellow areas of necrosis and a few small patches of round celled infiltration, but there was no evidence of malignancy. Differential staining did not reveal any sign of nervous tissue at any point. The tumour is therefore seen to be a fibroma of unusually large size. It may have started as a 'false neuroma' in the sheath of one of the segmental nerves close to the spinal column, but there is no longer any histological proof of this. Alternatively it may have arisen from a ligament or the periosteum of the spinal column.

I have taught and written hitherto that intrathoracic tumours arising from the thyroid gland can always be removed through the upper thoracic outlet. Nevertheless *Cases 2 and 3*, now to be recorded, illustrate conditions which render this impossible. In the first, there were dense adhesions in the neck and behind the sternum, and the tumour was placed deeply in the thorax. In the second, adhesions, seen in the x-ray photograph, were binding the tumour firmly to the aortic arch and to other structures in the neighbourhood.

Case 2—Dr Hector Mckenzie asked me to see this patient in consultation, and he has kindly written the following notes:

'The patient, a married woman, was sent to me by Dr Ind, of Sittingbourne, in June, 1911. She was then 42 years of age, and had had a swelling in her neck for four years. She had a moderate sized goitre, chiefly affecting the left side. It was smooth, and free from nodules. It seemed to extend behind the sternum. It was obviously producing pressure on the veins and on the trachea. The veins were prominent over the manubrium. There was some stridor, and shortness of breath and cough were induced by exertion. She was admitted to hospital.

'A first attempt to remove the goitre was unsuccessful. The left lobe of the thyroid was found to be much enlarged, to extend behind the sternum and to compress the trachea literally. The operation was followed by bronchitis and congestion of the base of the right lung. X-ray examination showed the trachea deflected to the right at the root of the neck and a shadow was seen projecting from behind the manubrium on both sides, but more on the left.

'A second attempt was made to remove the growth on Dec 1. The lower limits of the tumour could not be reached. Profuse hæmorrhage followed the endeavours and it was considered too dangerous to proceed further. Two and a half hours later the patient was blue, dyspnoeic, and unconscious, and Mr Max Pige, who was then Resident Assistant Surgeon, opened up the wound with the intention of performing tracheotomy. The trachea however could not be located and the symptoms being most urgent, the growth was seized between the fingers and pulled on, when part of it came away. More of the growth was then enucleated altogether. A mass the size of a large duck's egg was removed. As the dyspnoea was relieved tracheotomy was not performed. After this the patient remained very ill for some weeks with high temperature and rigors but she eventually was able to leave the hospital on Feb 3 1912.

'The laboratory report on the mass was that it was thyroid carcinoma. I think the subsequent history shows that this was not the case. The removal of part of the goitre relieved the patient for a time, and I did not see her again until Aug, 1917. Her principal difficulty then was a troublesome cough. The palpable part of the thyroid seemed only slightly larger than normal. There were still signs of intrathoracic pressure. In Oct 1921, I saw her once more. She was now a good deal worse. Cough had been very troublesome both in summer and winter. There was stridor and dyspnoea. The veins were very prominent over the upper part of the thorax, especially on the left side. It seemed to me that another attempt should be made to remove the intrathoracic goitre, but I felt a good deal of responsibility in advising the patient to undergo a third operation. She was very anxious to obtain relief.

When seen by me she was obviously in great distress. Every breath was laboured and breathing was only made possible at all by holding the head in a particular position in relation to the chest. A small adenoma of the thyroid could be felt on the right of the neck, but this was unconnected with the intrathoracic tumour. The tissues of the neck immediately above the sternum were matted together by the scarring of the previous operations. There was a close network of dilated veins all over the upper part of the front of the chest neck, and both arms. The face was cyanosed.

REMOVAL OF INTRATHORACIC TUMOURS

9



—Intrathoracic fibroma shown, where the capsule of the tumour was attached to the vertebra
(Natural size)

An x-ray photograph (Fig 8), taken by Dr Dudley Stone, showed a "large tumour in the superior mediastinum, reaching from above the clavicles down to the level of the seventh dorsal vertebra. It extended outwards for about 4 cm on either side of the sternum, backwards to the vertebral column," and forwards almost to the sternum. Although the tumour could be seen in the photograph to extend up into the neck, none of it could be felt or detected at this level. Operation afterwards showed that this part was entirely hidden behind



FIG 8.—Chest. Antero-posterior view. On the plate the trachea is seen encircling the right side of the tumour.

the trachea and oesophagus. The outline of the tumour was distinct from that of the arch of the aorta, which was pushed downwards and to the left. The trachea was displaced backwards and to the right and was narrowed antero-posteriorly as well as from side to side, that is to say it was compressed obliquely. The tumour descended below the bifurcation of the trachea. It was therefore, very deeply placed and the upper thoracic outlet was closed by scar

* At operation it was found to extend far back in the paravertebral space on the left side, as well as across the vertebral to the right.

tissues as by a lid. These conditions quite precluded any possibility of lifting it out in the ordinary way.

OPERATION, Nov. 24—As already seen, the tumour flattened and displaced the trachea, and extended beyond its division. There was also the possibility that even the slight irritation of the trachea produced by the introduction of a catheter might prove fatal. It was, therefore, not advisable to give an intratracheal anæsthetic, but to use an open method, the head being held throughout the operation in the position which the patient had proved for herself to be the only possible one. The anæsthetic was administered by Mr C. Langton Hewer. Mr Geoffrey Keynes assisted me at the operation.

The sternum was divided longitudinally as in the first case, but in this instance it was necessary to cut out along the third intercostal space on both sides in order to obtain sufficient access. The sternum was thus divided completely across (Fig. 9), and each side of the anterior thoracic wall was lifted up—opening double doors, as it were. Even when this had been done, it was only after the whole hand had been introduced into the chest from the lower end of the division in the sternum that the lower and hinder part of the tumour could be reached and lifted forwards. The upper part was buried in dense adhesions due to previous operations, these adhesions extending to well below the level of the upper border of the sternum. The tumour was separated and removed intact. The patient felt immediate relief and her convalescence was uninterrupted. She is now quite well.

The tumour was an elongated colloid adenoma of the thyroid, with rounded ends and an impression on one surface corresponding to the position of the arch of the aorta. It was 14 cm. long, 6 cm. thick, and weighed 307 grm. (11 oz.).

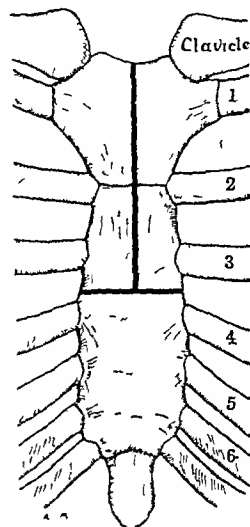


FIG. 9.—Case 2.
Line of division of sternum.

Case 3—The last case, Mr. M., a patient of Dr. Chapman, Beckenham, was a gardener, a very frail man, age 66, referred to me by Mr. F. Rose at St. Bartholomew's Hospital. This patient had apparently had an intrathoracic cystic adenoma of the thyroid for a long time. As in the former case, attempts had been made to remove it ten years before, but had failed. It was then drained and fluid material was discharged through a sinus for nine and a half years. Six months ago the discharge had ceased. Following on this, dyspnoea had commenced, and had become progressively more distressing. Admission to hospital had become a matter of urgency on account of the patient's rapidly increasing symptoms. The only possibility of relief was by the removal of the tumour.

In the former case the tumour had been entirely within the thorax, and there was no possibility of removing it through the neck. Generally, when the tumour is only an extension of an enlargement of the thyroid in the neck, it pushes its way down into the mediastinal tissues, which form round it an adventitious capsule. From this it may be shelled out with the greatest ease. Sometimes, however, a thoracic extension of this kind is not free below, and so cannot safely be lifted out from above, either with the fingers or with mechanical assistance.

In the present case the prolonged suppuration had given rise to chronic inflammation round the tumour and to firm adhesions. These were particularly well marked between the lower pole of the tumour and the arch of the aorta—the tumour very considerably overlying the aorta—and were seen in the roentgen photograph. At operation, April, 1921, the sternum was split, in this case under local anæsthesia in the usual way, and access to the tumour was good. It was, however, so firmly fixed by the adhesions at its lower pole that it could only be removed by dividing them partly by dissection partly by gently separating. The pulsations of the aorta could be felt directly under the finger all the time, and presumably there were other structures involved, equally important but less easily recognized.

* Since the above article was written this patient began to have pain and difficulty in swallowing. A roentgen examination showed a spherical opacity 12 cm. in diameter in the chest with its centre behind the articulation of the third costal cartilage with the sternum. At first I thought this was fluid distending the cavity from which the tumour had been removed. Exploration showed it to be neoplastic. The microscopic section having the appearance of spindle-cell sarcoma. Sections were then cut from the tumour removed on Nov. 24, 1921. This also was proved to be sarcoma. This is the only tumour removed by me which has not been examined microscopically at the time of removal, the reason being that it was encapsulated and an excellent specimen of a completely intrathoracic tumour, and it was desired to preserve it as such. The length of history—at least fourteen years—had seemed to exclude malignancy. The diagnosis in 1911 was carcinoma. The portions removed in 1921 and 1922 were undoubtedly sarcoma. The sections are available for examination.

It is possible that the 1911 tumour was of the nature of a follicular adenoma. It is difficult to believe that a carcinoma could have existed for that length of time. In this connection the article by J. P. Wilson of the Mayo Clinic in the *Annals of Surgery*, August, 1921, is extremely interesting.

Examination of the tumour after its removal showed that it consisted of a thick fibrous wall, in which no thyroid tissue could be found. It contained broken down debris. Cultures made from this showed no growth.

Dr. Trapnell writes me, Jan 25, 1922 that this patient is well as regards his thorax, and the breathing is quite comfortable. He has had a severe attack of nephritis since, and has osteoarthritis in one hip.

Any growth infiltrating within the thorax almost certainly cannot be removed, but it is not always possible to distinguish these growths from innocent tumours before operation. Even in dealing with innocent tumours the anatomical relations may be such as to make thorough exploration a matter of some danger. X-ray examination will show whether the margin is clearly defined, if it is, and if aneurysm can be excluded with reasonable assurance, there is the possibility that the tumour may be a fibroma, lipoma, dermoid cyst, goitre or hydatid cyst.

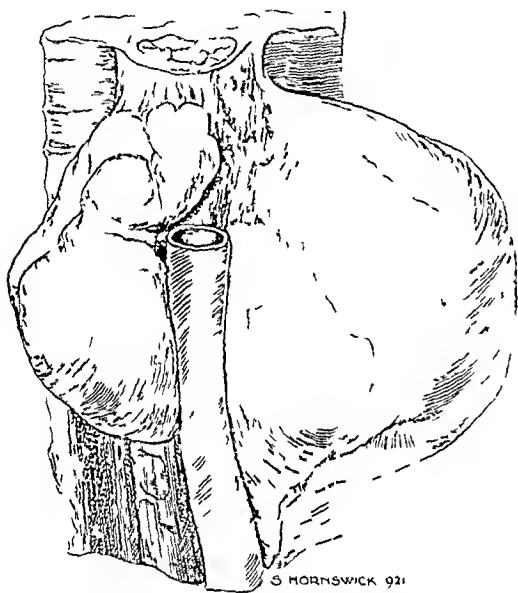


FIG 10—Fibromyxoma. Dr. Morley Fletcher's case.

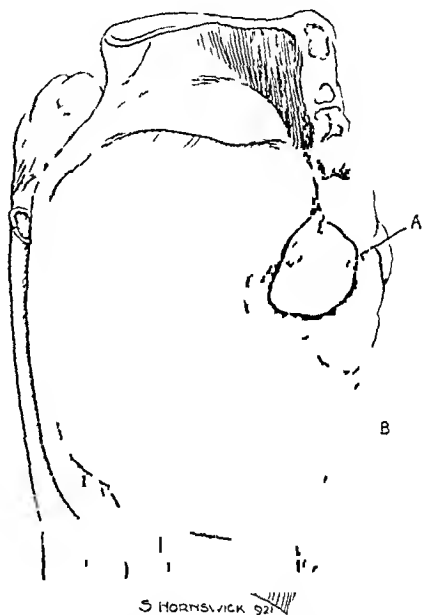


FIG 11—Section of specimen seen in Fig 10. A Site of origin of malignant growth (sarcoma). B The site of the growth is fibromyxoma.

It may be objected that there will be more danger to the patient in attempting to remove these, when large and deeply situated, than in leaving them alone. There are two main arguments against this view. (1) The dyspnoea, which in almost every recorded case has ultimately caused death, and (2) The possible occurrence of a malignant change in an innocent tumour. The first of these arguments is well illustrated by a case reported by Dr. Leopold.⁴ His patient, a man, age 37, suffered from a persistent cough of increasing severity. It was accompanied by shortness of breath and later, by pronounced dyspnoea. The physical signs and x-ray examination nine months after the onset of the symptoms showed that there was a tumour filling about four-fifths of the chest. There was no pain, difficulty in swallowing, alteration in voice, or loss of weight, but the slightest exertion produced distressing attacks of embarrassed breathing. Death, after about fifteen months' illness, was preceded by numbness of the arms, mental torpor, and, finally, a struggle for air. The necropsy showed that the thoracic cavity was almost filled by a lobulated mass which compressed the lungs against the vertebral column. This was found to consist of pale yellowish fatty tissue. It weighed 17 lb 6 oz, and measured 31 by 30 by 15 cm.

Although several cases of the other tumours mentioned above have been recorded, only four more cases of mediastinal lipoma could be found by Dr Leopold in the literature.⁴ Three of these ended fatally through increasing dyspnoea. The fourth, of the size of a tangerine orange, presented above the sternum, and was removed by Beatson of Glasgow. The seven fibromata recorded by Hare⁵ in the Fothergillian Prize Essay all ended fatally.

In relation to the second possibility mentioned, namely malignant change, Dr Morley Fletcher's case is interesting. The specimen (Figs 10 11) is now in St Bartholomew's Hospital Museum, and is described in the catalogue as "A large bi-lobed tumour of the posterior mediastinum, extending from the body of the sixth to that of the eleventh dorsal

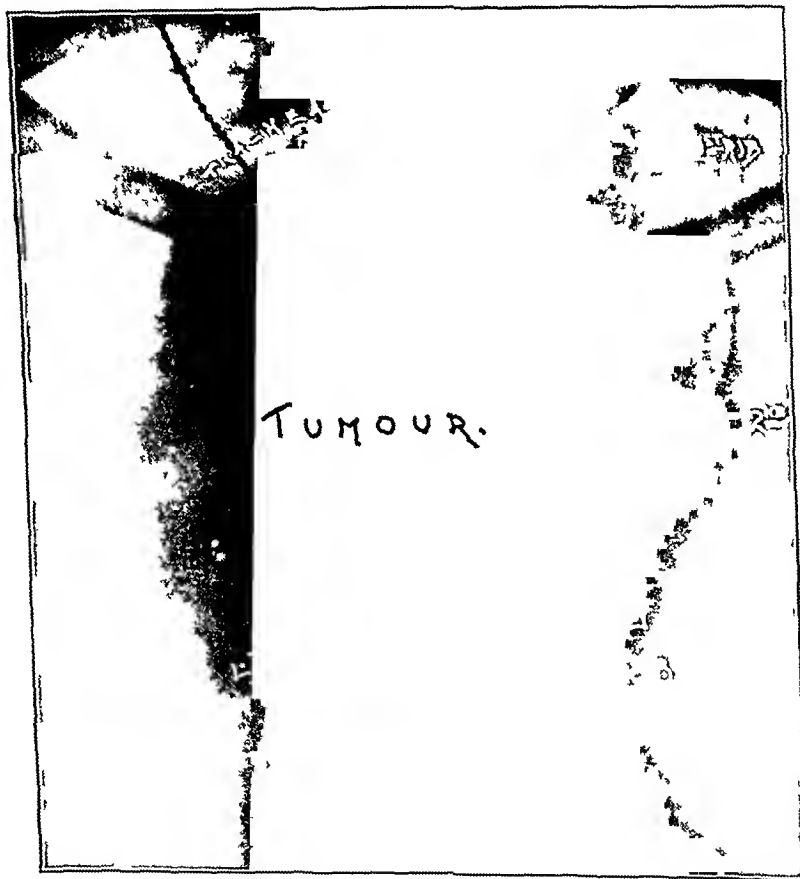


FIG. 12.—Aneurysm. The vertical limit is the same as the tumours in Cases 1 and 2.

vertebra. Lying behind the tumour on the left side are portions of the 7th, 8th and 9th ribs, which are invaded by a hæmorrhagic growth of totally different appearance. The bi-lobed tumour consists mainly of myxomatous and fibrous tissue with some nerve fibres and scattered groups of large round cells resembling sympathetic nerve cells. The hæmorrhagic part of the growth invading the ribs, and also that found in the bodies of the vertebræ, is a small spindle-celled sarcoma' (Specimen 2561B). The Committee on Morbid Growths (Hebb and Shattock) classed the primary growth as a fibromyxoma since they did not regard the presence of nerve tissues as an integral feature of the new formation. They believed the nerve-cells to belong to sympathetic ganglia which had become involved. It is reasonable to suppose that in this case the fibromyxoma had been in existence for a long time. Sarcomatous change occurred in it, at first in a small

area well marked off from the remainder of the tumour but invading surrounding structures later and ultimately killing the patient. It is worth noting the points of origin of this tumour and of the very similar growth recorded here in *Case 1*. In the present case the tumour apparently arose in, or in close proximity to, the sympathetic cord. In *Case 1* the only point of attachment seemed to be on the side of the body of one of the dorsal vertebrae, in close proximity to the sympathetic cord.

In another patient, a tumour having pathological characters exactly similar to those of the growth in *Case 1* blocked the outlet of the pelvis. It was 9 cm. in diameter, and prevented the delivery of a child, which Dr. Donaldson extracted by Cæsarean section. I afterwards removed the tumour, a retroperitoneal fibroma, and its point of attachment was seen to be at the first sacral foramen on the right side. These three tumours of identical structure had, therefore, analogous sites of origin along the vertebral column.

Sometimes it is very difficult to diagnose an aneurysm from other mediastinal tumours. An example of this is illustrated in *Fig. 12*. The patient suffered from symptoms almost identical with those of *Case 1* and of Dr. Leopold's case of massive lipoma. Dyspnoea on exertion was the only complaint. There was no pain at all and no discomfort as long as the patient took things quietly. For many months there was no expansile pulsation to be seen on the x-ray screen. The Wassermann reaction, however, was positive, and there was an indefinite tracheal tug. This induced those of us who were associated with the case to watch it carefully for a period extending over ten months. Then the dyspnoea increased. On screen examination the tumour appeared larger, and expansile pulsation was now obvious.

Fear of the unknown within the thorax has hitherto made us pause in dealing with tumours in this part of the body, even when the condition of the patient was distressing and dangerous. A greater familiarity is tending to remove our fear, but the last case recorded in this article emphasizes the necessity for an accurate diagnosis before a decision to explore finally prevails.

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LATE RESULTS OF MANIPULATIVE TREATMENT OF CONGENITAL DISLOCATION OF THE HIP

By E. LAMING EVANS, CBE LONDON

In reporting upon a series of 49 cases of congenital dislocation of the hip involving 61 joints, treated by me between 1903 and 1916 inclusive at the Royal and Royal National Orthopaedic Hospitals I am fully conscious of the many failures that have attended my earlier endeavours. The causes of these failures are in part general and in part individual. From 1903-10 I was largely influenced by the Lorenz technique which was in vogue at that time. With the meagre instruction derived from observing Lorenz reduce one case in London, I embarked upon the treatment of a deformity which had previously baffled surgeons whatever method they had employed. In 1903, orthopaedic surgery was in a very different condition from that in which we find it to day. The original British Orthopaedic Association was defunct and no new association had been formed. In America and on the Continent the opportunities for

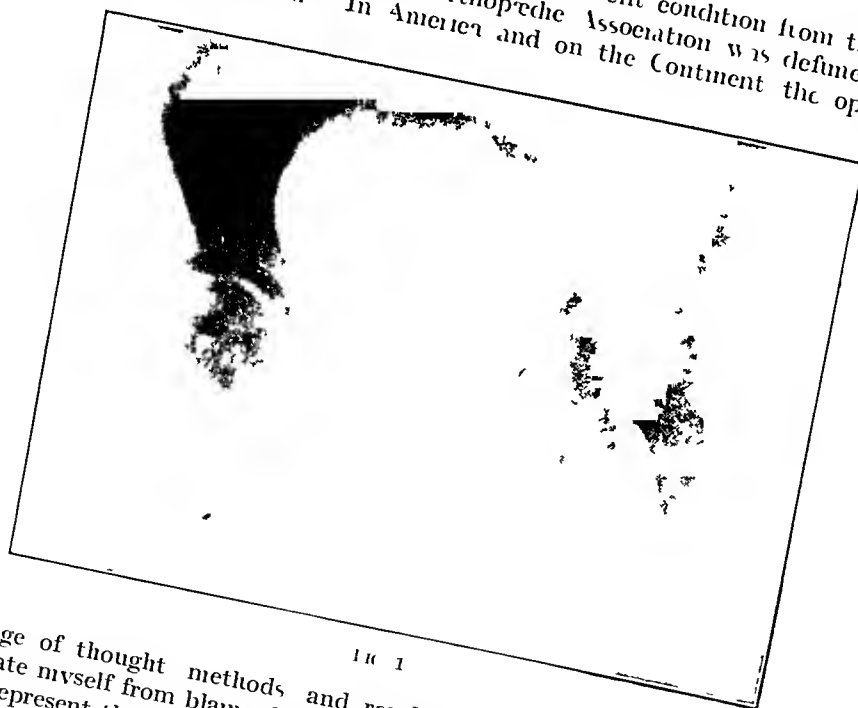


Fig. 1

the interchange of thought methods and results were well organized. I mention this, not to exonerate myself from blame but because the results of treatment in these 49 cases do not fairly represent the results which are now being obtained and by which the success of manipulative replacement should be judged. Further in my early cases no x-ray control was possible, for the reason that no x-ray installation was provided at the Orthopaedic Hospital at that time. From the experience thus gained, I am of opinion that no surgeon should attempt the treatment of a congenitally displaced hip unless he is in able to obtain x-ray proofs of his positions in plaster and of the subsequent growth of the acetabulum and upper end of the femur and unless he has had considerable opportunity of observing the methods and results of others. In this way only can the percentages of successes be increased.

Of the 49 cases, 45 were females and 4 were males. The left hip was dislocated 28 times, the right 9, and both 12 times—figures which show an unusually large number of left-sided cases. For the purposes of this paper I have endeavoured to re-examine all these cases within the last few months. The patients have been invited to attend the hospital and, if poverty has been pleaded, the railway fare has been offered as an inducement.

Eighteen cases, involving 23 joints, have failed to give me the opportunity of re-examination. I do not regard all these cases as failures. Some, no doubt, are, but others I observed for many years before the war, and showed radiograms of some of them at the British Medical Association meeting at Aberdeen in 1914, as cures. Of others again, I have had satisfactory reports from their medical attendants. But without recent x-ray confirmation they cannot be brought into any scientific classification, and I have therefore excluded from my list all cases that have not been recently examined.

As an example showing how an error might otherwise creep in, *Fig 13* represents a left unilateral dislocation in a female, age 24, ten years after reduction. It shows that four-fifths of the head only is covered by the acetabulum, whereas *Fig 14*, taken three years after reduction, shows the head completely surmounted by the acetabulum.

There remain for classification 31 cases, involving 38 joints. Classification so far has been simple, but further detailed classification of anatomical and functional results, as other observers have found, is difficult. The usual anatomical classification is divided into (1) Anatomical cures, (2)

Excentric reductions, (3) Anterior transpositions, (4) Relapses. Such may have served a useful purpose in the past, but is quite inadequate with our present knowledge. It came into being when surgical thought centred upon the dislocation as being the essential deformity, and the retention of the replaced head by the acetabulum as the highest ideal in the treatment. Such a classification is very one-sided and, though serving to describe the reaction of the acetabular elements to the stimulus of a replaced head, entirely ignores the reaction in the head and neck of the femur to the forces employed in reduction, and the stresses and strains of acetabular cohabitation.

CLASSIFICATION OF CASES

GROUPS	NO OF CASES	PER CENTAGE	AVERAGE AGE
1 Concentric reduction with normal head and neck	10	26.0	4.1
2 Concentric reduction with changes in the head and neck	13	34.0	5.1
3 Excentric nearthrosis	5	13.0	4.5
4 Anterior transpositions	4	10.5	4.8
5 Posterior dislocations	1	2.5	6.5
6 Loss of head and neck	1	2.5	2.9
7 Unreduced dislocations	4	10.5	6.75
Total Cases	38	—	—

In examining the radiograms of late results, I have been struck by the infrequency of anatomical cures as evidenced by them. By a strict definition an anatomical cure is one which an x-ray examination shows is indistinguishable from a normal hip. To refer to two points only—a normal acetabulum shows a double contoured roof, after reduction of a congenital dislocation, this double contour is of the rarest occurrence. I do not think I have seen it more than a few times. If we allow this variation, and such a change as the persistence of some mammillation of the roof to be within the normal then the percentage of anatomical cures materially increases. Though we may reasonably include these and some other minor changes as being within the normal limits of anatomical variation, the grosser changes in the head and neck of the femur that occur in a large proportion of so-called anatomical cures would seem to demand a class of their own.

For this reason, I consider the above classification, which is employed for my cases, to be more satisfactory.

GROUP 1.—Of 38 joints recently examined, 10 only showed concentric reductions with normal heads and necks, whilst 13 showed concentric reduction with changes in the heads and necks, making 60 per cent of concentric reductions, but only 26 per cent of anatomical cures.



FIG 15



FIG 16

Fig 15 represents a case of concentric reduction with normal head and neck. It is from a left unilateral case of a girl, reduced at the age of 4, x-rayed nine years after reduction. Function of the joint is complete.

Fig 16 shows the opposite side for comparison.

Fig 17 is a case of a girl with bilateral dislocation, reduced at the age of 4 years 8 months, and re-examined eight years after. The horizontal direction of the epiphyseal cartilage will be noted, and the entrance of the inner and lower angle of the neck into the acetabulum. It would appear that this horizontal direction of the epiphyseal cartilage has protected the head from displacement, and that the projecting angle of the neck has acted as a buttress preventing slipping of the head.

Fig 18 shows a left unilateral dislocation in a female, reduced when 4½ years old, and x-rayed nine years later, which shows a concentric reduction, with normal head and neck with a crescentic epiphyseal line.



FIG 17



FIG 18

CONGENITAL DISLOCATION OF THE HIP 19

Fig 19 is from a case of left unilateral dislocation in a female, age 7½, and shows the result twelve years later. The acetabulum is shallower than normal, but the



FIG 19

head and neck are well formed and the reduction is concentric. The functional result is excellent.

Fig 20 represents the result five years after reduction of a left unilateral dislocation in a patient, age 3½ years. This case had been reduced and kept in plaster-of-Paris

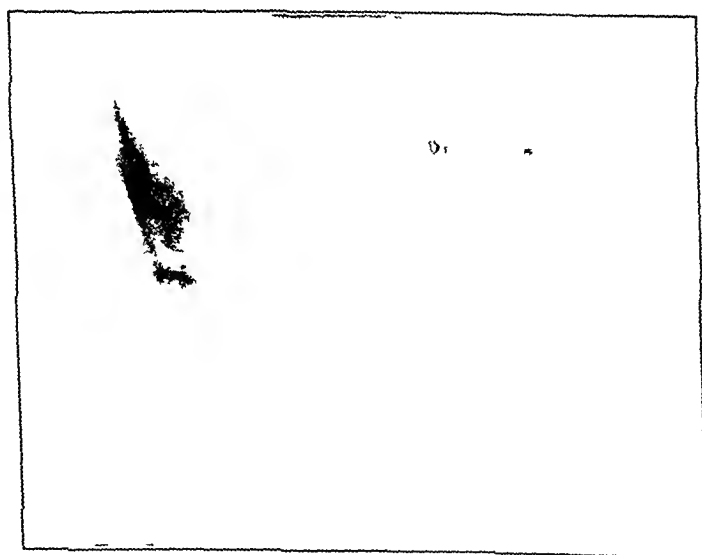


FIG 20

elsewhere, but retention failed. Fibrosis of the adductors necessitated their tenotomy before re-reduction could be obtained.

GROUP 2—Turning to the concentric reductions with changes in the head and neck, a great variety of pathological lesions is found. The commonest is a diminution of the angle of inclination, and this is present in nearly all cases. Further changes are buffer-shaped heads, and flattening and spreading of the capital epiphysis over the neck, absorption of the neck, and, less frequently, an increase in the angle of declination.

Fig 21 is from a bilateral case in a female, reduced at the age of 7 years 2 months. Radiogram taken six years later. The left side shows a concentric reduction with *coxa vara*, the right an eccentric ne-



FIG 21

arthrosis with void neck and atrophy of head and neck.

Fig 22 shows a buffer shaped head on a shortened neck, from a bilateral dislocation in a boy, reduced at the age of $6\frac{1}{2}$ and x-rayed thirteen and a half years later.

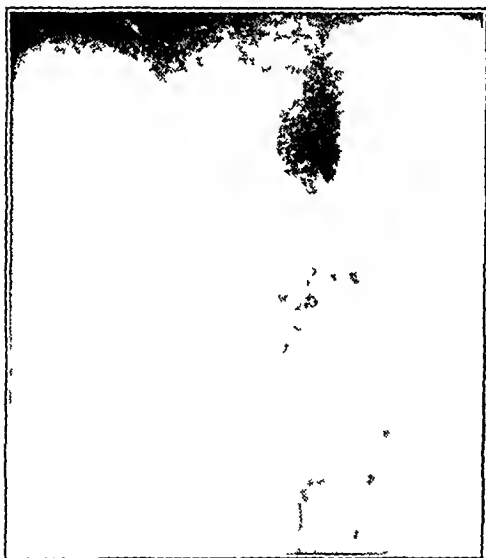


FIG 22

GROUP 3—In the eccentric nearthrosis cases are included those which show changes in the acetabulum. These changes consist of an absorption of the upper part of the acetabulum, so that the femoral head forms a new joint within the limits of the original acetabulum but not concentric with its centre. Changes in the head and neck almost invariably accompany the changes in the acetabulum, and for the most part consist of a partial absorption of the head and neck. The changes are similar to those occurring in a dry arthritis, but there is no evidence to suggest that they are of tuberculous origin.

Thus, Fig 23 represents a case of a female, age $5\frac{1}{2}$, left unilateral, x-rayed thirteen



FIG 23

years after reduction, which shows an excentric nearthrosis with well-shaped head directed at an angle of 130° to the shaft of the femur, with almost complete absorption of the neck. The upper end of the femur is bent laterally in the trochantene region

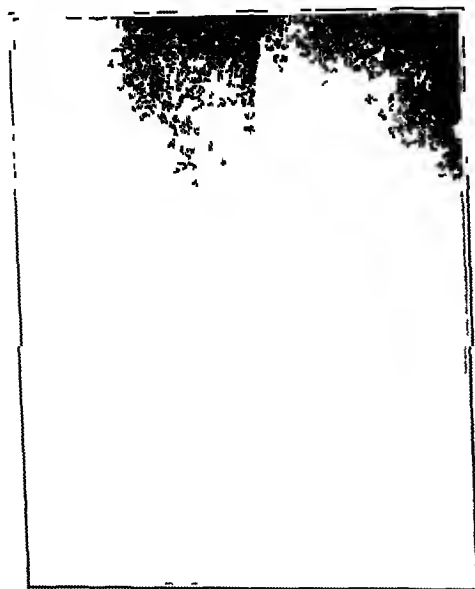


FIG 24



FIG 25

Compare with this *Fig 24*, a bilateral case of a female, age $8\frac{1}{2}$, x-rayed nine years after reduction. The right hip, here represented, was reduced easily, the left gave much trouble. The right shows a coxa vara of severe degree and much shortening of the neck, but the head is well formed (? Place this in *Group 2*, i.e. concentric reductions with changes of head and neck)

Fig 25 is a left unilateral dislocation in a female, age 5, x-rayed ten years after reduction. It shows an excentric reduction with shortening of neck, without alteration of the angle of inclination, and with a well-formed rotund head. The neck is constricted about its centre.

Fig 26 is from a left unilateral dislocation in a girl, age $4\frac{1}{2}$, x-rayed eight years later. There was marked coxa valgus when reduced. One year and

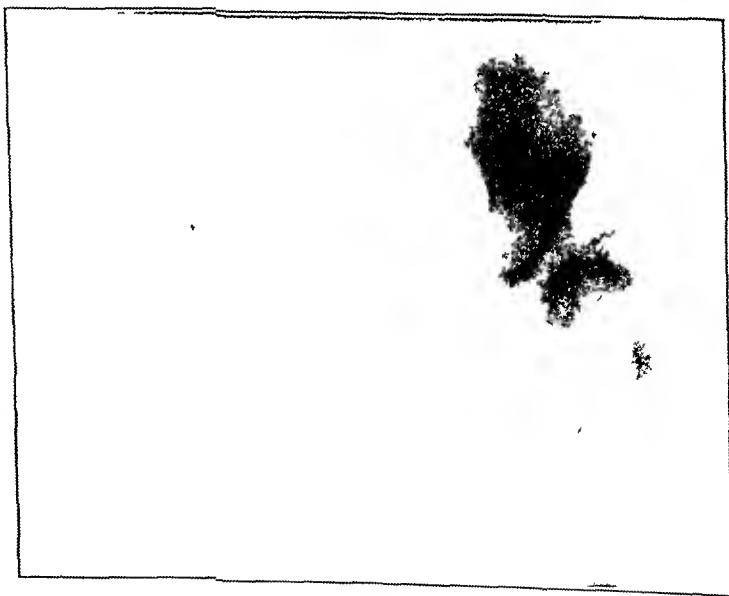


FIG 26

half later the hips were symmetrical. There was then interrupted observation. Now the radiogram shows an excentric nearthrosis with high valgus and the capital epiphysis displaced outwards.

GROUP 4—With reference to anterior transpositions, I have noted few changes in the head and neck, which probably accounts for the extraordinarily good functional results

GROUP 5—Posterior redislocations conform in behaviour to the untreated cases, except that changes in the length and direction of the neck are common



FIG 27

Fig 27 is an example bilateral dislocation, boy, reduced at 6½ years. Radiogram taken thirteen years later. The right femoral head is bullet-shaped, the neck much shortened and at an angle of 100°. Clinically, extension is short by 15°, but flexion is complete

GROUP 6—Complete loss of the head and neck is rare. I have one case only to record, a right unilateral, and a radiogram is given in *Fig 28*. The patient was a boy age 2½, with multiple deformities. The illustration was taken sixteen years after reduction. Unfortunately I have no radiogram of the original condition. Notwithstanding the loss of the head and neck, the lateral apposition of the side of the femur to the pelvic wall affords a stable joint. He plays football, as he says, better than most, and is very much pleased with the result. Except that there is no scar I should have guessed that he had wandered to another's care and had his head and neck excised. *Fig 29*, which represents

his opposite hip, shows considerable contraction of the centre of the neck, with an expanded head

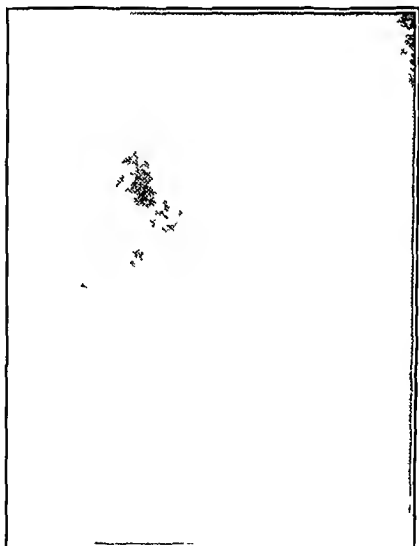


FIG 28



FIG 29

Fig 30 is from a girl, age 10½ years, in whom I fractured the neck, treating her by extension with a long Liston. The radiogram gives the appearance eleven years afterwards—a posterior dislocation, without obvious change in the angle of inclination. She is certainly no worse than she would have been if nothing had been done

Of the functional results it is much more difficult to speak, because the personal element largely enters into the question

No orthopædic surgeon can be successful unless he is an optimist, and it is very difficult to dissociate optimism from after-results. Further, it is essential that any classification that is to be intelligible to the ordinarily retentive memory should be short. Hence, for statistical purposes we are compelled to adopt such a classification as good, fair, or bad.

In general terms —

1 Concentric reductions without any changes in head and neck are good. In fact, I think they are very good.

2 Concentric reductions with changes in head and neck vary, some are good, others fair. I have to record one bad result in this class—an ankylosis in external rotation and abduction with coxa vara of 90° .

3 Eccentric reductions depend upon the degree of absorption. Three cases show good functional results and two fair. It is still too early to say whether progressive arthritic changes will not vitiate these early satisfactory results. I am not optimistic on this point.

4 Anterior transpositions give good functional results as a rule.

5 Posterior redislocations are indistinguishable from untreated cases.

I wish to record one case which showed every prospect of a concentric reduction with normal head and neck, which was attacked with anterior polyomyelitis of the same limb, and an eccentric nearthrosis resulted.

My method of reduction was founded upon Lorenz's technique—gradually forces were diminished and muscles spared, so that my later cases were reduced by a much more gentle and less disruptive process than the earlier ones. Except in very few cases where I have used internal rotation after Lange's method of retention, I have employed a short spica reaching from the waist to above the knee. Early locomotion on a high patten has been adopted. In bilateral cases the period of retention in plaster has been shortened as much as possible—from three to six months. In unilateral cases I have kept up retention for much longer than is usually accepted—eighteen months to two years. With few exceptions retention has been maintained in 90° flexion, 70° abduction, and an indifferent rotation. Weindorff's axillary abduction has promoted successful retention in difficult cases where the acetabular roof has been markedly deficient.

After removal of the plaster, abduction in walking has been secured by applying a $1\frac{1}{2}$ -in. patten to the sound side.

I have attached great importance to a plaster bed made according to the formula 90, 70, 0, up to the end of the third year after successful retention. In unilateral cases one hip only has been enclosed in the plaster. Exceptional cases have been treated with modifications according to the structure and stability of the joint, but, in general, the above may be taken as routine.

I have fractured some necks, but no femoral shafts. I have had no nervous or arteriovenous complications. There has been one tragedy—the death of a patient, age 8 $\frac{1}{2}$, after reduction, from double pneumonia, without a post-mortem examination.



FIG. 30

LATE RESULTS OF TREATMENT OF CONGENITAL DISLOCATIONS OF THE HIP

By H A T FAIRBANK, DSO, OBE

IN opening the discussion on "The Late Results of the Treatment of Congenital Dislocation of the Hip" at the meeting of the British Orthopaedic Association at Liverpool in December last, I reported the results of my personal experience with cases treated before the war. The subject seems to be of sufficient importance to warrant the publication of a more detailed paper than it was possible to present before the above meeting. If any excuse were needed it might be found in the fact that even at the present time there are surgeons who have still to be convinced that the affection is curable, while the numbers of cases left untreated till an age has been reached when completely successful treatment is impossible are far too large. I do not propose to discuss the reports published in medical literature, as I think the space at my disposal will be fully occupied in dealing with the results of my personal experience, small though it is.*

The Committee of the Association decided that the discussion should be limited to results noted after a lapse of at least five years from the date of operation. In presenting this report I propose to refer to some of the complications met with during treatment, and to consider the influence, if any, exerted by these complications upon the results. The cases investigated include all those treated by me, at Ormond Street and elsewhere, before the war, that is, during the years 1903 to 1914 inclusive. The number of cases amounts to 146, with a total of 175 hips. Every effort has been made to be honest in this investigation with the same object in view all disasters met with in this series are recorded.

Method of Treatment—The method of treatment adopted has been the Lorenz manipulative reduction as a rule, though in some difficult cases every conceivable manoeuvre has been tried. Only in the early cases was the Lorenz method followed strictly, before long the amount of violence used was reduced considerably, unnecessary damage to the skin over the adductors was particularly avoided. Muscles were ruptured only when stretching was insufficient. The skin was sterilized as for an open operation. In every case the manipulations after the reduction had been accomplished, suggested by Lorenz, with a view to getting the head well home in the acetabulum, were carried out. These I believe to be a very important part of the operation. A small sandbag, except in the earlier cases, was used as a fulcrum behind the head of the femur in place of the Lorenz wedge. The hamstrings were usually left alone till later, they were gradually stretched while the leg was in plaster. The Lorenz position was chosen for fixation in plaster, the leg being retained in this right angle position for never less than six months. Cases with particularly poor stability were fixed in the 'axillary position' for a few months and then brought to a right angle. A change of position during treatment in plaster—for instance, internal rotation—was only adopted when the head of the femur showed a tendency to ride forwards. Patients were encouraged to walk after the first month in plaster, while active and passive extension of the knee was also encouraged. The knee was not included in the plaster as a routine. In the younger children the plaster was removed at the end of six months and nothing further done. In the older children—particularly in bilateral cases—the plaster cast would be re-applied once or more often with diminished abduction, while later, massage, exercises, and passive stretching by hand and weight in the direction of hyperextension, would be carried out for some months.

* It was originally intended that this report should include all the cases treated at the Hospital for Sick Children, Great Ormond Street up to 1916. In spite of the courtesy and generous assistance of my colleagues, I have been able to do so little in the way of investigating the present condition of their cases that I am not yet in a position to offer any report on them.

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Thirteen cases were treated by open operation during the period under consideration. The results of these operations are not included in the tables of results given below, though most of the cases are recorded in those tables as failures after manipulative reduction or attempted reduction. The open operations will be dealt with separately.

Anatomical Results—As a result of consultation with some of the other members of the Association, the cases have been divided into three age groups, namely (1) Under three years, (2) Three to six years, (3) Six years and over. Only 7 cases were above nine years of age. The oldest was fifteen. No case has been omitted.

Table I shows the total number of hips dealt with and the results recorded in all those cases in which treatment was completed. This list gives some details not included in the next table, and assists in explaining how the latter was compiled.

Table I—RESULTS IN TOTAL NUMBER OF HIPs TREATED BY MANIPULATIVE REDUCTION, OR ATTEMPTED REDUCTION UP TO THE YEAR 1914

Unstable (Open Operation) Hips in which stability after reduction was so poor and prognosis so bad, that open operation was deemed advisable. *Sepsis* Suppuration in the hematoma of the adductors. *Lost sight of* Those known to have died from intercurrent disease before final removal of plaster cast, and those who ceased to attend before the treatment was complete and in which the result is unknown.

Age	Failed to Reduce	Unstable (Open Operation)	Sepsis	Lost Sight of	Femur Fractured	Cures	Anterior Reposition	Relapses	TOTALS
Unilateral —									
Under 3	—	—	1	4	—	26	3	2	36
3, 4, and 5	1	1*	1	5	1	26	6	3	44
6 and over	4 + 1*	1*	—	3	2	9	8	—	28
Bilateral —									
Under 3	—	—	—	—	—	8	5	2 + 1*	16
3, 4, and 5	4*	—	—	1	2	14	7	5 + 2*	35
6 and over	—	1*	—	1	—	4	4	4	14
TOTALS	10	3	2	14	5	87	33	19	173

* Open operation. Two other cases were treated by open operation after Lorenz' by other surgeons.

Table II—LATE RESULTS OF MANIPULATIVE REDUCTION

The hips in columns of *Cures* and *Anterior Repositions* were traced for at least five years after reduction. The *Failures* include every hip treated in which the result was known to be a failure. The figures are derived from columns *Failed to Reduce*, *Unstable (Open Operation)*, *Sepsis*, *Femur Fractured*, and *Relapses* in Table I.

Age	Class 1 Cure	Class 2 Cure	Anterior Reposition 1	Anterior Reposition 2	Failures from all Causes	TOTALS
Unilateral —						
Under 3	10	4	3	—	3	20
3, 4, and 5	8	1	2	4	7	22
6 and over	1	5	2	5	8	21
TOTALS	19	10	7	9	18	63
Bilateral —						
Under 3	6	1	1	1	3	15
3, 4, and 5	5	—	1	3	13	22
6 and over	—	4	1	2	5	12
TOTALS	11	5	6	6	21	49

In *Table II* are included only those cases—comprising 112 hips in all—in which the result is known after a lapse of at least five years from the date of reduction. All the known failures shown in *Table I*, from whatever cause arising, are included in this table e.g., failure to reduce, fracture of the femur, relapse, etc.

The vast majority of those reported as cures or 'anterior repositions' have been followed up for a much longer period than five years, as may be seen by a glance at *Table III*. All cases classed as 'cures' have been proved by x rays, with the

Table III—LENGTH OF TIME FROM REDUCTION TO DATE OF FINAL EXAMINATION AND REPORT

Years after reduction	5	6	7	8	9	10	11	12	13	14	15	17
Number of hips	3	6	8	12	4	7	4	15	2	9	2	1

More than half were seen over 10 years after reduction more than three quarters over 8 years

exception of one. This case was known to be a cure at first and was examined by me more than twelve years after reduction, and I am quite sure that the hip was perfect in every way, anatomically and functionally, the legs were equal in length, no complaint of any kind could be elicited, repeated attempts at obtaining a skilgram have so far failed. Among the 'anterior repositions' are included 6 which were known to be



FIG. 31.—Case 1. Congenital dislocation of left hip in a girl, age 1 year 10 months. The radiogram taken 10 years after reduction showing a Class 1 Cure. Function perfect.

'anterior repositions' and not cures before the five-year limit was reached, but of these radiograms could not be obtained at a later date although their present functional result can be reported. The results other than failures have been classified as 'anatomical cures' and 'anterior repositions', each class being again subdivided. The radiograms will indicate better than words the type of case in each class. I suggest that this classification might be worthy of general adoption, as I submit that it is sufficiently elaborate without being too complicated. In the Class 1 Cures are included those cases where

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the head of the femur is in the acetabulum and approaches the normal in size and shape (Figs 31, 32, 33, 34, etc) Among the *Class 2* Cures are placed those which show an equally definite anatomical cure, but in which the head of the femur is distinctly abnormal, either in the direction of being mushroomed (Fig 35), varoid (Fig 36), or of being partly or completely worn away by what I prefer to call 'absorptive arthritis' (Fig 37, etc) By 'anterior reposition 1', I mean a case with a fairly well-formed rounded head opposite the upper lip of the acetabulum, with or without the formation of a socket at this point (Figs 38, 39, 40) In 'anterior reposition 2' of this group are placed the cases with gross changes in the bones, which usually take the form of flattening and absorption of the head of the femur, and flattening and condensation of the neck of the femur, poorly-

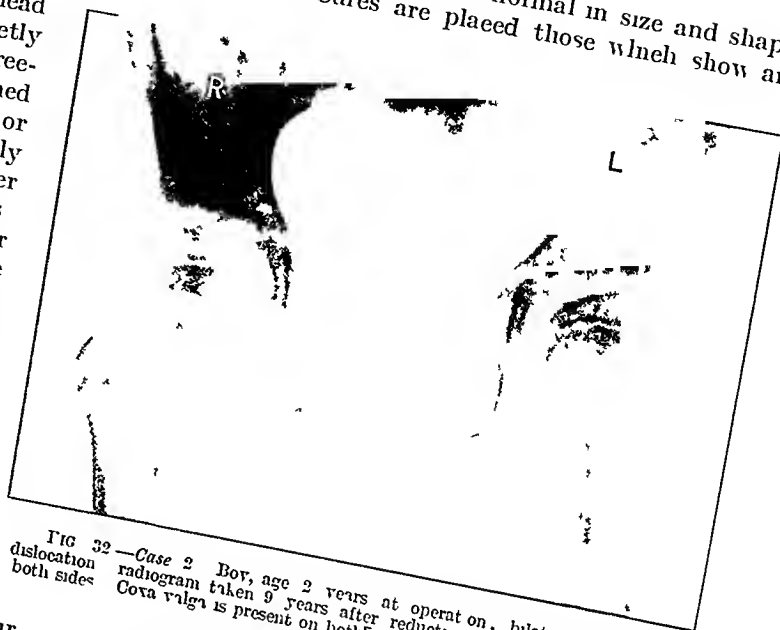


FIG 32—Case 2 Boy, age 9 years at operation, bilateral congenital dislocation radiogram taken 9 years after reduction showing *Class 1* Cure Coxa valga is present on both sides, but function is perfect

No doubt some surgeons would classify separately the hips showing such deformities as coxa vara, marked anteversion of the neck of the femur, poorly-

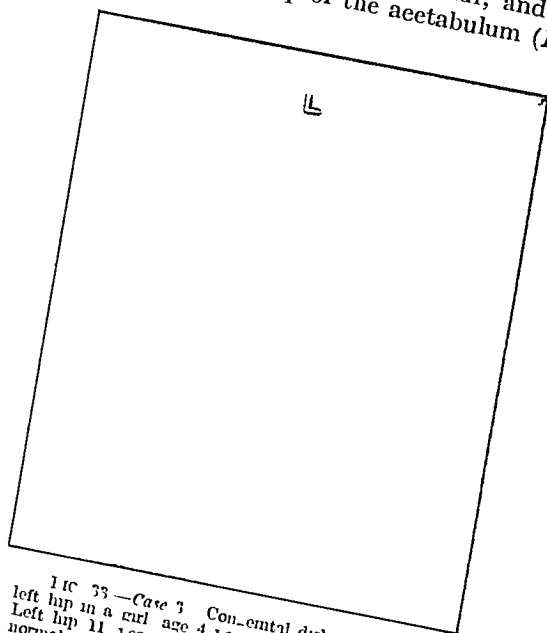


FIG 33—Case 3 Congenital dislocation of the left hip in a girl age 4 years at time of operation Left hip 11 years after showing result sufficiently normal to be called *Class 1* Cure i.e. lowest limit of first-class cures The acetabulum is still poorly developed and head of femur is imperfectly covered by upper lip of acetabulum Function perfect

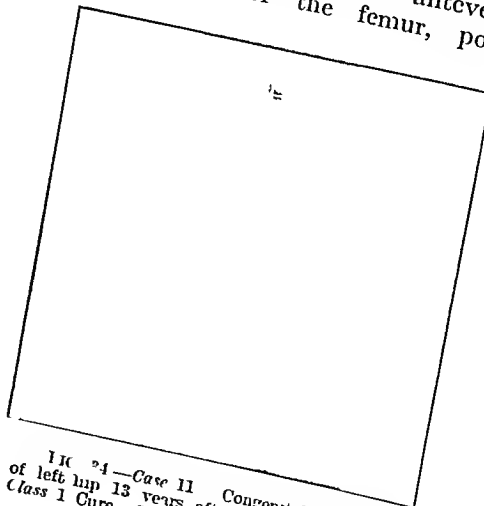


FIG 34—Case 11 Congenital dislocation of left hip 13 years after reduction showing *Class 1* Cure Function perfect

Table II shows the percentage results at the various ages of the unilateral and bilateral cases These are worked out from the figures given in Table II, they even therefore I developed acetabulum, etc, in the late radiogram, but in any attempt to standardize the estimation of late results, over-elaboration seems inadvisable

think, be taken as on the low side since all the known failures and disasters are included whereas only those partial or complete successes which have been examined after a lapse

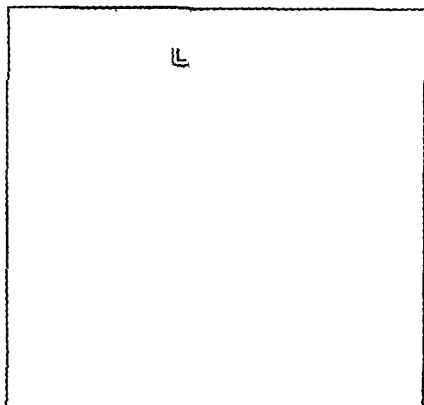


FIG 31—Case 4. Congenital dislocation of left hip in a girl, age 1 year 10 months. The radio-gram is taken nearly 1½ years after reduction shown, Class 2 Cure. Head of femur is mushroomed. Radio-graphic changes suggestive of pseudo-coxalitis well seen during treatment. A radio-gram taken before operation showed total absence of ossific centre for dislocated head of femur this centre being present on the normal side. Function perfect.

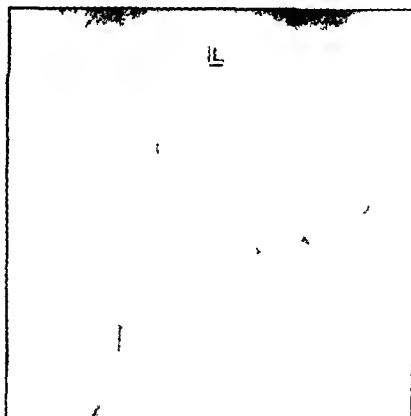


FIG 36—Case 12. Boy, age 6½ years. Congenital dislocation of left hip, with some coxa vara. Radio-gram taken 10½ years after reduction showing Class 2 Cure. Marked coxa vara. Function perfect.

of five years are included. It will be noticed how, in the unilateral cases, the percentage of cures falls rapidly as we pass from the younger to the older children, while the per-



FIG 37—Case 5. Girl with dislocation of the left hip, age 10½ years at time of operation. The hip 11 years after reduction shown, Class 2 Cure. Gross changes are seen in head and neck of femur as result of absorptive arthritis. Function is good but not perfect.



FIG 38—Case 6. Girl, age 6½ at operation. Congenital dislocation of right hip 13 years after reduction. Result, anterior reposition, Class 1. Head of femur fairly rounded. Function fair.

centage of 'anterior repositions' increases. In the bilateral cases the figures are somewhat surprising, though again showing the advantage of early operation. I think the reason

for the cases of six years and over showing a better percentage result than those in the middle age-group lies in the fact that the former were subjected to a more careful selection,



FIG 39—Case 7. Girl, age 1 year 11 months with bilateral congenital dislocation. Radiogram taken 10 years after reduction of both hips. Result: anterior reposition, Class 1, on both sides. The right hip is more displaced than the left, but opposite the upper lip of the acetabulum. Function fair.

while in the latter reduction was attempted in almost every case seen. At any rate, these tables serve to prove yet again the enormous advantage a young child has over one

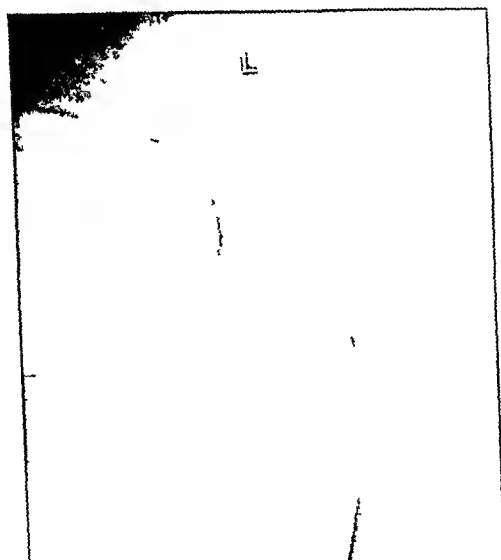


FIG 40—Case 10. Left hip 11 1/2 years after reduction showing anterior reposition, Class 1. This hip was not treated by open operation. Function fair. Compare with Fig 39—the right hip of same case.

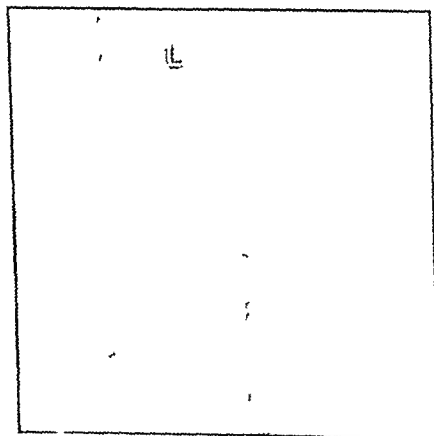


FIG 41—Case 8. Girl, congenital dislocation of left hip, age 8 years at operation. Radiogram taken 13 1/2 years after reduction of the dislocation. Result: anterior reposition, Class 2. Gross changes in the head and neck of the femur with flattening and condensation of bone in the region of the upper acetabular margin the result of absorptive arthritis. Function good.

whose treatment has been unwisely delayed. It is generally agreed that bilateral cases are more difficult and troublesome in every way than the unilateral, and no one expects as good results in the former as in the latter.

Table IV—PERCENTAGE RESULTS AT VARIOUS AGES COMPILED FROM TABLE II
(FIVE YEARS AND UPWARDS AFTER REDUCTION)

Unilateral			Bilateral		
Age	Cures per cent	Anterior Repositions per cent	Age	Cures per cent	Anterior Repositions per cent
Under 3 years	70	15	Under 3 years	46.6	33.3
3, 4 and 5	40.9	27.2	3, 4 and 5	22.7	18
6 and over	28.5	33.3	6 and over	33.3	25
All ages	46	25.3	All ages	32.6	24.4

Function—Yet another table (*Table V*) is presented, showing the functional results arranged under the headings 'good', 'fair', and 'bad'. The vast majority of 'good' are absolutely perfect—that is to say, the walk is excellent, and no complaint whatever is made, included are a few who limp if very tired or who tire a trifle sooner than they should, although at other times no fault can be found with the function of the joint. No attempt is made to define exactly what is meant by 'fair' and 'bad', since the personal element cannot be excluded from any such rough classification. It will be seen that these small figures suggest that the functional result in most cases agrees with the anatomical result.

Table V—FUNCTIONAL RESULTS OF CASES INCLUDED IN TABLE II
(FIVE YEARS AND UPWARDS AFTER REDUCTION)

AGE	Class 1 Cure				Class 2 Cure				Anterior Reposition I				Anterior Reposition II			
	Total	G	F	B	Total	G	F	B	Total	G	F	B	Total	G	F	B
Unilateral —																
Under 3 years	10	10	—	—	4	3	—	1	3	1	1	1	0	—	—	—
3, 4, and 5	8	7	—	1	1	1	—	—	3	1	2	—	2	—	1	1
6 and over	1	1	—	—	5	4	1	—	2	1	1	—	5	3	1	1
Bilateral —																
Under 3 years	6	5	—	1	1	—	—	1	4	—	2	2	1	—	1	—
3, 4, and 5	5	5	—	—	0	—	—	—	2	—	1	1	3	—	1	2
6 and over	2	2	—	—	2	1	—	1	1	1	—	—	2	—	—	2

G = good F = fair B = bad

The *Class 2 Cures* are not associated with such good function as the *Class 1* while the 'anterior repositions' give even worse results. It is only occasionally, in my experience, that the anatomical and functional results markedly disagree.

It is about the later histories of the imperfect anatomical cures such as I have put in *Class 2*, and of the 'anterior repositions' that we particularly want information. Are we justified in thinking that we have done permanent good to the patient when the result can only be placed in one of these latter classes? Although in many we have undoubtedly improved the function greatly, do we really delay the onset of pain and increasing disability, i.e., arthritis, which I think we rightly regard as the inevitable fate of the untreated case? This is one of the points on which more information is so urgently needed. Time alone can solve the problem, but I think we have to admit that in some cases reduction—even successful reduction—has been followed by changes in the joint which the operation was intended to prevent. This knowledge should influence us, I think, very strongly.

when we attempt to raise the age limit for reduction. My own feeling is that results do not warrant attempts at reduction being made after nine years of age in a unilateral case, and six years in a bilateral. Of 8 cases treated when over nine years of age, only one shows a cure, and that is a *Class 2*. In 4, attempts at reduction failed, while in one of these a greenstick fracture of the femoral neck was produced.

It is interesting to note that in four unilateral cases with a *Class 1* Cure, the affected leg is now the longer of the two, the difference varying from a quarter to half an inch. These four were all operated upon before the age of three. The final examinations were made eight to ten years after reduction. In the *Class 2* Cures there is one, aged two years at operation and examined twelve years later, with half an inch of lengthening, in this case the function is bad, owing to arthritic changes in the joint, while the 4 cases in *Class 1* have excellent functional results. The lengthening seems to involve the tibia and fibula as well as the femur. In one 'anterior reposition', a girl $6\frac{1}{2}$ years at the time of reduction and now aged 19, there is a quarter of an inch of lengthening. The tibia on the affected side is half an inch longer than that of the normal leg. In some other cases with imperfect results the changes in the head and neck of the femur suggest the presence of a greater amount of shortening than is actually found by the usual method of measurement. In only two of the unilateral cases with a *Class 1* result is there any shortening of the affected limb.

It is noteworthy that only a quarter of the 'anterior repositions' are credited with good functional results. In a quarter the function was 'bad', while in the remaining half it was 'fair'. At least a quarter complain of pain in the hip, and more than half the cases limp. Shortening amounts to about one inch in the cases seen from 6 to 14 years after operation. Two, age 8 and 9 years respectively at the time of operation, showed as much as 2 inches of shortening 13 and 11 years later. Lordosis was absent in some, present in many, and varied from a slight to a severe degree. Lordosis should, I think, have been avoided in a greater number of cases, if the after-treatment had been more efficiently carried out. Trendelenburg's sign was present in 11 cases, absent in 9, indefinite in 3, while it was not noted in the remaining 4. Those with a positive Trendelenburg had on the average a worse functional result than those without this sign. These details are given for what they are worth, because there seems to me to be some grounds for thinking that the general opinion on 'anterior repositions' inclines towards optimism.

Age—Some surgeons seem to be against reduction before the age of two years. Theoretically the earlier the reduction is made the more likely is an anatomically normal joint to result. The writer is inclined to operate as early as 18 months in the absence of any indication for delay. In this series, 9 cases were operated upon between the ages of $1\frac{1}{2}$ and 2 years. The results show 7 cures, 5 in *Class 1* and 2 in *Class 2*, and 2 'anterior repositions', one in each class. Better results than these are not likely to be obtained by delaying the operation. The results obtained when reduction has been delayed till after the ninth year are in marked contrast to the above, and have been referred to already.

Sex—As regards any possible influence of sex on the result, it is only necessary to say that reduction was not, on the average, more difficult, while the results were rather better, in boys than in girls.

Early Prognosis and Late Results—By testing the stability of the reduced hips at the time of operation, and comparing the prognosis thus determined with the late results, it has been found that such prognosis can be made with fair accuracy, especially in the younger children.

COMPLICATIONS

We now pass to the consideration of some of the complications of treatment.

[*Note*—Wherever figures are given below, these invariably refer to the number of instances of a particular complication met with in the 112 hips dealt with in this report, and not in the total hips treated up to 1914.]

Complications of the Operation itself—Fractures of the femur occurred on five occasions. This figure gives a misleading impression as to the frequency of this accident.

five is the total number of fractures met with in the writer's experience, i.e., in just over 200 operations. No fracture has occurred in the last 97 attempted reductions. In each case of fracture all attempts at treatment of the dislocation were abandoned. So far as is known no permanent harm resulted from this accident.

The only instance of injury to a nerve was one in which the sciatic was bruised in a prolonged and unsuccessful attempt to reduce a dislocated hip in a girl, age 14, the Lorenz wedge being used. Fortunately the nerve recovered completely. A sandbag has been used instead of the wedge ever since, while attempt at reduction at such an age has not been repeated.

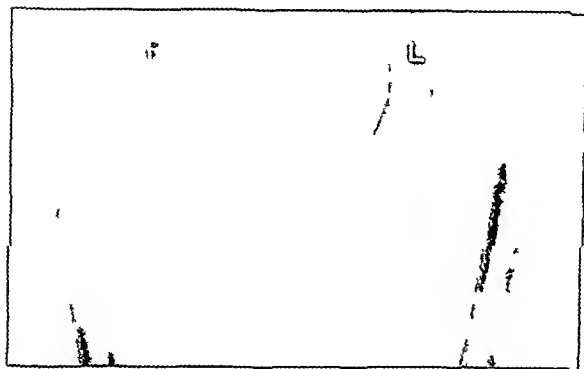


FIG 42—Case 10. Boy, age 2 years. Bilateral dislocation before operation.

Suppuration of the hematoma caused by damage to muscles occurred twice. The details of the first case were reported in 1908*. Treatment by another surgeon had been followed by relapse, and, probably as a result of the former operation reduction was extremely difficult for the age (3½ years). Infection of the hematoma occurred, and in spite of radical surgical treatment it ended fatally. The second case is recorded below. It is advisable to be cautious in dealing with cases that have relapsed after previous efforts at cure, while care should be taken to see that a

child is in good general condition and free from any source of infection before attempting reduction. Unnecessary injury to muscles is to be avoided, not only because of the risk of possible infection, but because the ultimate functional result may be affected thereby.

Pseudo coxalgia (osteochondritis deformans juvenilis)—Changes in the head of the femur similar to those seen in this affection though absent before operation, were noted after reduction in 7 cases. The fluffy, broken up appearance of the capital nucleus was more obvious than the flattening typical of coxa plana. One case had bilateral dislocations but changes in the head of the femur were found on one side only (Case 10, Figs 42-45). Another similar bilateral case with pseudo coxalgia in one hip only has been met with, but as it could not be followed for so long as five years it is not included with the seven. In only one of the seven, the bilateral case, had any special difficulty, involving excessive trauma, been met with during reduction. Their ages ranged from 20 months up to 7 years at the time of operation. The changes in the head of the femur were noticed

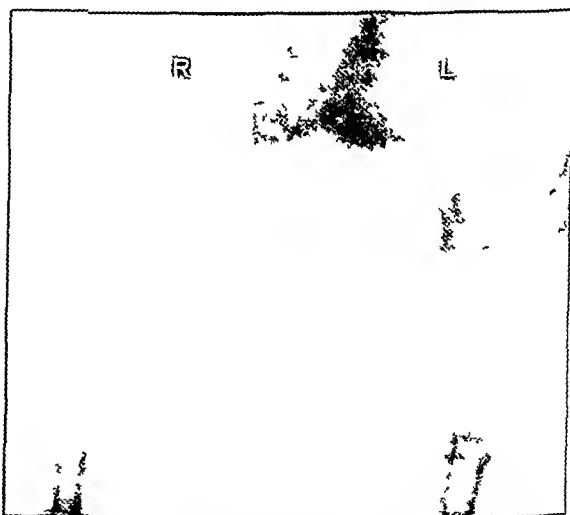


FIG 43—The same case as Fig 42, one year after reduction, showing slight changes in the head of the right femur. Note the upper lips of the acetabulum.

as a rule about twelve months after reduction, but this must not be taken as an indication of the exact time of onset of the affection. Hospital cases were x-rayed a few days after reduction, and if all went well the examination was not repeated until the affected limb had come down parallel to its fellow or nearly so. The late results of these seven cases show 6 cures (4 with good function, 1 with fair, 1 with bad) and 1 'anterior reposition' (function fair). In all but one, be it noted, the late radiogram shows a flat and spread-out or mushroomed head, placing them in the second classes of the two groups. It would seem therefore that this affection, whatever its true nature, does not lead to relapse of the dislocation, but does mar the result to some extent.

Arthritis—Stiffness of the hip, suggesting subacute or chronic arthritis, coming on in the course of treatment was experienced six

times. The stiffness was first noticed from 7 to 15 months after reduction, and lasted for periods varying from a few weeks to 18 months. The exact date of onset of those



FIG 44—The same case as Fig 42, but 3 years after reduction showing typical fragmentation (pseudo-coxalgia) of the right hip. No changes on the left side.

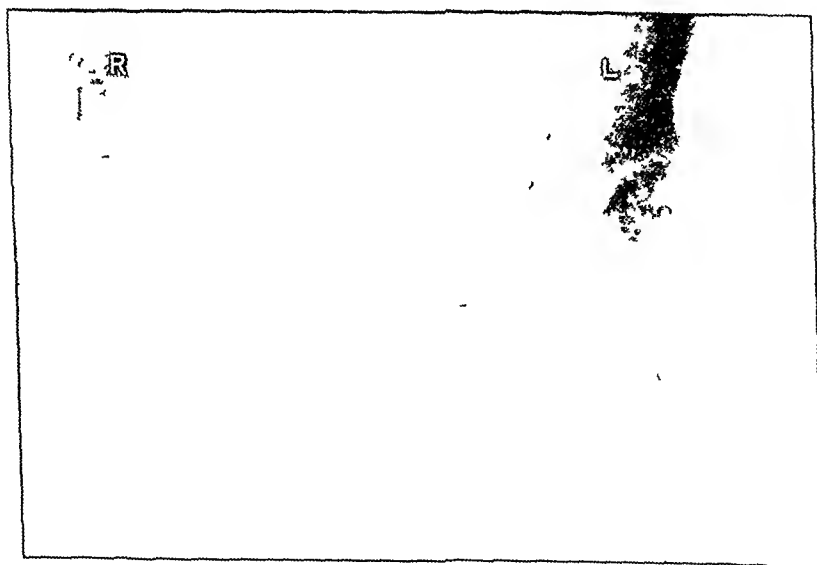


FIG 1—The same case as Fig 42 12 years after reduction. Results: Right, Class 2 Cure with mushroomed head. Left, Class 1 Cure. Gait perfect, but walks very little on account of pain.

discovered soon after removal of the plaster is necessarily doubtful. It occurred in children of all ages (2½ to 9 years). In one case a girl age 9, it followed a fall ten months after reduction; in another, changes resembling pseudo-coxalgia were seen in the skingrams. The duration of symptoms varied from a few weeks to 18 months, but in

every case the stiffness gradually subsided with rest, very often imperfect rest. The number of cases is too small for definite conclusions to be drawn, but it would seem that the results are spoiled to some extent. These cases showed only one *Class 1* Cure three *Class 2*, and two 'anterior repositions', one in each class. Only 3 showed good functional results. This arthritis is quite distinct from 'absorptive arthritis' already referred to as a rule the former is not followed by the changes in the joint which are characteristic of the latter.

Anterior Displacement—Displacement forwards of the head of the femur during treatment is, of course, not a very rare complication. This usually takes place while the child is in the plaster case, but may occur later. The head of the femur was found riding forwards on the horizontal ramus of the pubis in five cases. In the majority of these the head was easily restored to its normal position by bringing the knee a little forward and rotating in the femur and re-applying the plaster. In only two was any difficulty experienced in correcting the displacement, both these and one of the others resulted in 'anterior repositions', while the remaining two were 'cures'. If left uncorrected this complication will certainly diminish the chances of a cure, but if looked for, and corrected without delay when found, it has only a small effect on the results. The above figure (5 cases) does not give a correct idea of the frequency of this complication, for 17 cases (19 hips in all) were met with in the total pre-war series. By chance only five of these could be traced for 5 years and upwards, but the rest, as far as is known, give results similar to those recorded above.

Ankylosis—I have met with one case of ankylosis after the so-called 'bloodless' reduction in this series. In this, a unilateral case of 2 years with easy reduction, the hæmatoma of the adductors suppurred, pyæmia followed, both hips became secondarily infected and both ankylosed. One hip in a girl of 6 years treated by open operation gradually became fixed, though aseptic throughout. Trans-trochanteric osteotomy was performed to get the limb into better position, three months later (2½ years after reduction) a definite though small amount of movement was present in the hip-joint. Unfortunately the final result in this case is unknown.

Vulnerability to Infection—Have we any evidence that a congenital dislocation reduced or unreduced, is more liable to infections than a normal hip? We have the cases of arthritis following reduction, but these may well be traumatic in origin rather than infective. In the first of the two cases just mentioned the infection attacked the normal as well as the affected hip, in addition to various other portions of the body. Open operation is, I feel sure, more likely to be followed by an apparently aseptic arthritis leading to ankylosis than is manipulative reduction. I have one or two cases that seem worthy of brief mention here.

A girl had her right hip reduced at the age of 2½ years. The hip, 4½ years later, was 'm' and perfect in every way, the leg being slightly the longer of the two. This happy condition was said to have continued for another 7 years, i.e., 11 years in all, when the girl suddenly got pain in the leg after an attack of appendicitis. A diagnosis of tuberculous disease was made by another surgeon, and a single Thomas hip splint was worn for eighteen months. When seen 3 years later the hip was almost fixed, and was painless. X rays showed a curious condition, the head of the femur being divided into two and covering the upper part of the neck, which latter projects inwards below to articulate with a socket at the site of the upper lip of the acetabulum. One can only classify it as an 'anterior reposition'. It is probable that this hip was never a perfect 'cure' and very gradually passed into the condition of 'anterior reposition', and that some low-grade infection of this subluxated joint took place. The only early radiogram available, taken 13 months after reduction when re-examined in the light of a wider experience, suggests that the joint was not quite so stable as we then thought it to be. The infection was certainly non-tuberculous.

A bilateral case, age 2 years, cured on both sides, had an attack of subacute rheumatism with cardiac lesion in 1918, i.e., about 9 years after reduction. All affected joints cleared up except the hips, which continue to give him so much pain that he

hardly walks at all. Yet his gait is extremely good, and x rays show the hips to be *Class 1* and *Class 2* Cures respectively (*Case 10*). If pseudo-coxalgia is inflammatory in origin, as Dr. Calve has, I think, proved, the cases with this complication cited above must be considered as instances of infection attacking the hip after reduction.

Lastly, there is the painful condition of the hip-joint, with increasing flexion and tendency to adduction, which, it is generally admitted, sooner or later affects all cases in which the dislocation has not been treated. The pain, etc., are, I think, due to an arthritis attacking the abnormal joint. Though it is highly probable that successful reduction, particularly when this is accomplished at an early age and results in an anatomical cure approaching the perfect, will free the joint of this tendency to arthritis which is otherwise inevitable, at present we have no data from which to draw conclusions. Many more years must elapse before we can say that a *Class 1* Cure will stand the test of age and other factors as well as a hip that has never been dislocated. Attention has already been drawn to the need of information on the fate of those hips which can only be placed as *Class 2* Cures or 'anterior repositions'. It is at least doubtful in these cases whether we have really staved off the arthritis which would have attacked them sooner or later had nothing been done.

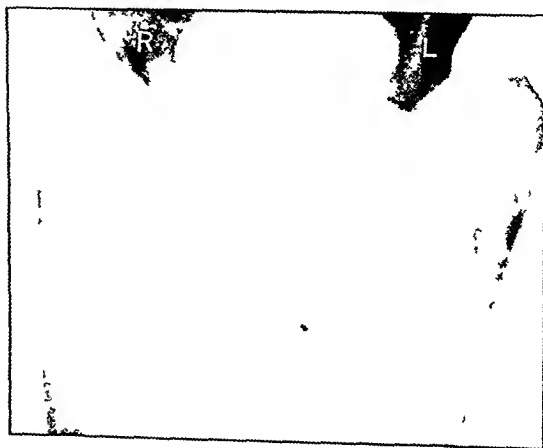


FIG 46—*Case 13*. Girl, age 2 years 10 months
Bilateral dislocation. Before operation.

Traumatic and Late Re-dislocations.—There are two cases of sudden re-dislocation—one case the result of a fall—in this series. One, a girl, age 4½ years at the time of the

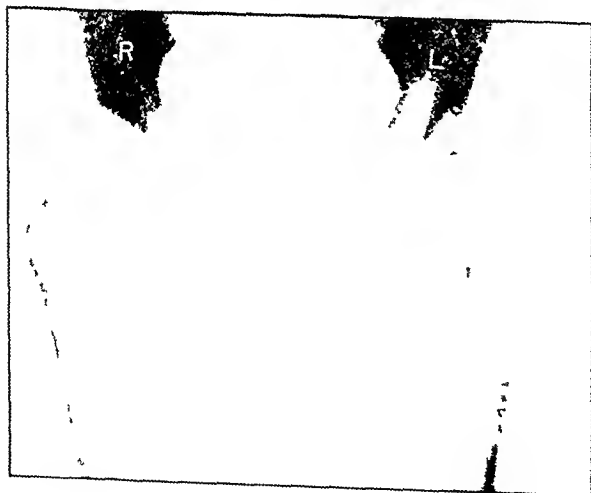


FIG 47—The same case as Fig 46. 2½ years after reduction showing cure. Acetabular upper lips still good.

reduction, was apparently cured when last seen 4 years after operation. A fall resulted in sudden re-dislocation 9 years after reduction. Mr. Bankart, who saw her and reduced the dislocation, tells me the hip seems to be ankylosing. (This case is classed as a failure in my tables.) In another case, a girl of 5, the hip was 'put out' 7 months after reduction, i.e., soon after removal of the plaster. There was no definite fall to account for the dislocation. Reduction and fixation for a further 8 months had resulted in a *Class 2* 'anterior reposition'.

Late re-dislocations without apparent cause are not common. Most hips which seem to relapse late have really been imperfect from the first. I have had however two cases

which were true though partial, late relapses. One was a 'cure' after 2½ years, the upper lip of the acetabulum being unusually well developed but now, after 11 years, it is an anterior reposition. The other a bilateral case, age 2½ years, showed a double cure with well-formed upper lips after 2 years. The hips are said to have been perfect for 10 years after which the left began to give trouble and this is now an 'anterior reposition', while

the right is still cured (*Figs 46-49*). Two other cases were thought to be cured 2 to 3 years after reduction, and are now 'anterior repositions', but they were not at first the stable-looking joints seen in the two previous cases

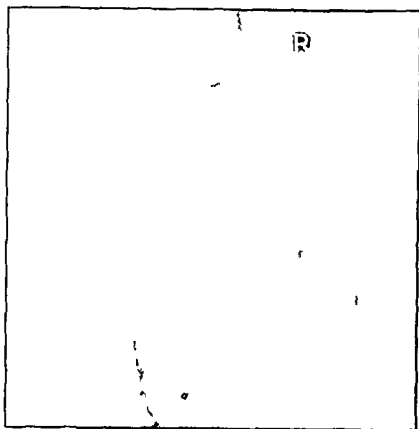


FIG 48—The same case as Fig 46, showing right hip 11 years after reduction. Class 1 Cure with poor acetabular upper lip and unstable looking joint. Function perfect

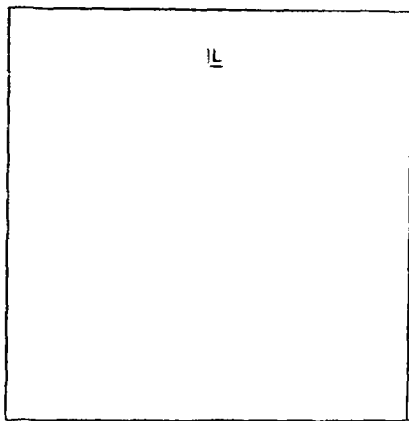


FIG 49—The same case as Fig 46. Left hip 11 years after reduction showing anterior reposition, Class 2. Function fair

Open Operation—There were 13 operations in the pre-war cases. Nine open reductions were attempted after manipulation alone had failed to reduce the dislocation or had been followed by relapse. In two of these reduction was not accomplished, while a third was so unstable that the attempt at a cure was abandoned. Infection occurred in one, which eventually ankylosed above the acetabulum. Unfortunately I have not been able to trace a single one of the remaining five cases, though two were known to be cures for 2½ and 4 years respectively, a third was 'in' though the joint was fixed by fibrous ankylosis as reported above, and one was an 'anterior reposition'. In three others in which the hip is reduced by manipulation was unstable, a small opening was made into the joint to verify the reduction, and then an attempt was made to fashion an upper lip for the acetabulum, while in a fourth case an upper lip was made without opening the joint. Only two of the four have been traced, of which one relapsed and one is a Class 1 Cure (*Fig 50*, see also *Fig 40*).



FIG 50—Case 13. Girl bilateral dislocation. Both hips reduced at 3½ years. Right hip treated by open operation and formation of an upper lip to the acetabulum. Radiogram of right hip 9½ years later showing Class 1 Cure with good acetabular margin. Function excellent

CONCLUSIONS

The following conclusions seem justified

1 If cases are sent to the surgeon sufficiently early, i.e. before the end of the third year, an anatomical cure ought to be obtained by manipulative reduction in something like

75 per cent of the unilateral and 50 per cent of the bilateral cases. In the vast majority of these the function should be excellent, at any rate for many years

2 Of those giving an imperfect result by the manipulative method, some additional 'cures' should be obtained by a second manipulative reduction, followed in two or three weeks' time by open operation, the object of which is the making of an upper lip for the acetabulum without opening the joint (This I regard as a much less grave operation than open reduction)

3 Open reduction should not be necessary in the younger cases but is specially to be considered in those cases of the middle age-group in which manipulative reduction has proved impossible. Only in carefully selected cases should this operation be performed after the age of 6 years.

4 After reduction full right-angle abduction should be maintained for a minimum of six months.

5 Although some of the cases with an 'anterior reposition' show remarkably good functional results, we must, in the present state of our knowledge, hesitate to attribute to these patients any permanent advantage over the untreated cases.

6 Physical treatment after removal of the plaster case, in all but the youngest patients, may exert a permanent influence on the function of the hip, but it probably has no effect on the anatomical result.

Finally, I wish to express my gratitude for the care and trouble taken by Dr Robert Knox and Dr Bertram Shires over the radiograms.

ACUTE PHLEGMONOUS GASTRITIS

By CHARLES J. MACAULEY, DUBLIN

THIS rare affection of the stomach is usually described as occurring in two forms, either as a circumscribed submucous abscess or as a diffuse purulent infiltration of the submucous coat. The localized variety appears to have been first noted by Varandaeus, as far back as 1620, while the earliest description of the diffuse type is attributed to Andral in 1839. In 1910, J. E. Adams recorded a case due to the pneumococcus, and in the same year Leith wrote the first comprehensive account of the disease to appear in English, in *Allbutt and Rolleston's System of Medicine*. Since 1910, no case, so far as I am aware, has been recorded in Great Britain or Ireland, but several papers have appeared in America in recent years, notably those of Rixford and Novak, while Sundberg has detailed 215 cases, including 17 from the clinics of Stockholm and Upsala.

It is said that a similar condition may occur in the duodenum, and Sherrin (*Choyce's System of Surgery*, 1915) quotes Ungeimann as having recently collected six cases of phlegmonous duodenitis, in three of which the disease was localized in the duodenum alone.

Phlegmonous gastritis may occur at any age, but is most common between 20 and 60, and is said to be much more frequent in men than in women.

Etiology—Adams' case was due to the pneumococcus, but all the others in which bacteriological examination was made have been due to the streptococcus, generally in pure culture, but sometimes in mixed infection with the *Bacillus coli*.

In considering the possible portal of entry, the cases may be conveniently divided into two groups—

a SECONDARY CASES—These form a small group in which an obvious lesion is present from which the suppurative process spreads, e.g., malignant or callous ulcer or an operation wound. Thus, it has been known to follow gastro-enterostomy and gastrotomy. The path of invasion in such cases is clear.

b PRIMARY OR IDIOPATHIC CASES—In these there is no discoverable lesion of the mucous membrane. Here we can only assume, in accordance with present-day conceptions, that the organism gains entrance through some minute abrasion of the mucous membrane, or is carried by the blood-stream from some distant focus. That entry takes place through the mucosa in the majority of cases is generally held. Thus, in all of Sundberg's personally observed cases there was a previous history of chronic gastritis and in my own case dyspepsia had been present for some years. A previous history of chronic alcoholism is said to be frequent, but recent cases, at any rate, afford very little evidence of such a connection.

Against the view that the infecting agent enters through traumatic or other lesions in the gastric epithelium, Shatara quotes the experiments of Symmers, who failed to produce the lesion in animals by feeding them on ground glass and inoculating streptococci and pneumococci by way of the blood-stream and stomach tube. That this evidence is by no means convincing, however, is shown by the work of Simmons and von Glahn who, after a series of careful experiments with ten dogs, conclude 'that the ingestion of ground or powdered glass produces no lesion either gross or microscopic, in the gastro-intestinal tract of dogs.'

That the path of infection may be by way of the blood in some cases would seem clear from the association which has been frequently noted of gastric phlegmon with conditions in which one might reasonably assume the presence of a blood infection.

Dittrich (quoted by Brinton) observed many cases of suppurative gastritis in 1851 during an epidemic of puerperal fever in Prag, and noted the frequent occurrence of erysipelas in such cases. In Lehnhoff's patient the abdominal attack followed immediately on what was believed to be an influenzal sore throat. Three of Rixford's cases occurred in the winter of 1916-17, during which an unusual number of severe streptococcal infections, especially sore throats, occurred in San Francisco. In a large group of cases, however, there has been no discernible lesion of the gastric mucosa and no primary focus has been found elsewhere, and in these so-called idiopathic cases it is commonly assumed that the micro organisms gain access through some very minute breach in the mucosa. There is no record of blood cultures having been made in any of the cases, probably because the diagnosis has been made, at the earliest, only on the operation table, and few cases have survived more than a few days thereafter. In any case, as Rixford points out, positive findings would be of little value, since they might be secondary to the stomach lesion. In my case certainly there were all the clinical signs of a severe septicaemia. If one were to consider the origin of gastric phlegmon in the light of modern views on similar and allied conditions, the possibility would seem to be not too remote that the vast majority of idiopathic cases are of hæmatogenous origin. It is believed—and the belief is supported by considerable evidence—that infection of the wall of the gall-bladder is commonly derived from the blood-stream, whether portal or systemic, and the pathological picture in phlegmonous cholecystitis resembles in many ways that of phlegmonous gastritis. Rosenow has isolated streptococci from the bases of gastric and duodenal ulcers, and claims to have established by animal experiment their power of elective localization in these and other regions, and the primary foci, frequently in teeth or tonsils, may present no gross and obvious lesion, and are thereby liable to be overlooked. Of interest also in this connection is the work of Reeves on the gastric and duodenal blood-vessels which indicates that the vessels in the submucosa of ulcer regions are longer, smaller, and have fewer anastomoses than elsewhere, thereby predisposing to thrombosis. "Since the vessels are more liable to be occluded by emboli, it is reasonable to suppose that they are an important factor in the production of ulcer by hæmatogenous infections." Gastric phlegmon appears to affect primarily the region which is so commonly the seat of peptic ulcer, since the circumscribed submucous abscess is always located at the pylorus, while the early cases of the diffuse type have all been limited to the same region. It is tempting, therefore, to assume that gastric phlegmon—which is almost always of streptococcal origin—may start in the same way as gastric ulcer, by hæmatogenous deposit, with the difference—of degree only—that the former, especially in its fulminating types, is due to a much more virulent infection, and one which is probably aided and accelerated by a diminished local and general resistance.

Pathology—The suppurative process—having once started in the submucosa, generally at the pyloric end—may spread widely through the submucous tissue or may be localized to form an abscess, the extent of spread depending on the virulence of the infecting organism, and on the presence or absence of induration of the stomach wall which might be expected to act as a barrier. Thus, phlegmon starting in a growth or ulcer appears to be less fulminating in its onset and development than the primary cases.

The possible terminations of the process are —

1 Perforation through the mucosa into the gastric lumen, with conceivable recovery. While such cases have been recorded, the correctness of a diagnosis based on pus in the vomit is open to considerable doubt.

2 Perforation through the serosa, with resultant peritonitis, or extension of the infection through the serous coat, without actual rupture.

3 Death from toxæmia or septicæmia before rupture can occur. In 33 per cent of Sundberg's fatal cases nothing was found to indicate peritonitis.

4 Encapsulation forming an abscess simulating a neoplasm as in Novak's case.

It is interesting to note that, even in the diffuse forms, the suppurative process never seems to spread beyond the pyloric ring or cardia.

The naked-eye appearances of the diseased organ are given in the account of my own case

Symptomatology—In the majority of cases of the diffuse type the clinical picture is pretty definite. In the fully developed stage, the features are (1) Sudden onset of intense epigastric pain, (2) Vomiting, early and persistent, but not recurrent, (3) Marked prostration, (4) Fever, which often rises to 104° , (5) Epigastric tenderness and rigidity, often to left of middle line, (6) Leucocytosis (10,000 to 20,000—mainly polymorpho nuclear), (7) Often congestion at the base of one or both lungs, (8) Dry brown tongue, intense thirst, often hiccough, and sometimes a tender mass in the epigastrium. The symptoms rapidly merge into those of profound and progressive peritonitis, which frequently precedes the end. Death takes place in three to ten days.

Diagnosis—Novak says that in only a few instances (Chvostek, Dorbeck, McCaskey), not all of established authenticity, is diagnosis said to have been made during life. Rixford made the diagnosis at operation by inserting a hypodermic needle into the stomach wall and withdrawing pus—a plan which has much to recommend it when the appearances of the stomach are suspicious. In my own case the picture of the stomach at operation was so striking that I had no hesitation in making the diagnosis, although my anaesthetist and assistant were frankly incredulous. In attempting to make a pre-operative diagnosis the following conditions have to be considered—

1 *Basal Pneumonia*—This is often closely simulated by the high fever, hurried respiration, and dullness at one or both lung bases. In cases of doubt Rixford advises exploratory laparotomy under local anaesthesia as being less dangerous than waiting, and as possibly the only certain means of making a diagnosis by exclusion at a sufficiently early period.

2 *Hæmorrhagic Pancreatitis*—May be indistinguishable, but in pancreatitis fever is commonly absent. In a recent case of hæmorrhagic pancreatitis under my own care the temperature had been persistently subnormal, a feature which, combined with a rapid thready pulse and deep epigastric tenderness without rigidity, enabled me to make the diagnosis with a fair degree of confidence, which was justified by the operative findings.

3 *Perforation of a Gastric Ulcer*—Resembles phlegmonous gastritis in its sudden onset, but differs from it in being followed almost at once by muscular rigidity over a widely increasing area, and by the absence of high fever. The terminal stages of both affections, being those of general peritonitis, are, however, likely to be indistinguishable.

4 *Acute Phlegmonous Cholecystitis*—Here also the sudden onset and high fever show a close resemblance, but the symptoms in cholecystitis are right-sided, and the distended gall-bladder may be palpable. In my own case it was the only condition I could think of as fitting the clinical picture, though the absence of tenderness and rigidity in the gall bladder region was quite definite. Only in pneumonia is an exact pre-operative diagnosis necessary, since the other conditions demand immediate surgical intervention in any case. The disease so seldom comes within the domain of the practical surgeon, that unless he has previously met with such a case, he is unlikely to make any other diagnosis than that of some urgent condition in the upper abdomen which requires exploration.

Treatment—When one considers the frequently fulminating nature of the disease, and the difficulties in the way of early diagnosis, it is not surprising that the records of operation in this condition are gloomy in the extreme. There are recorded only three operative recoveries in authentic cases, and these are to the credit of Bovee, Koenig, and Novak. The cases of Bovee and Koenig were of the diffuse variety, but limited to the pylorus, the operative procedures being incision and drainage by rubber tube in the former, and partial gastrectomy by the Kocher method in the latter. Novak's case was a localized abscess involving the pylorus and simulating a tumour; recovery followed partial gastrectomy by Balfour's modification of the Polya method. On ordinary surgical principles incisions into the stomach wall would be indicated, but such a procedure could scarcely afford adequate drainage in the diffuse type where both walls may be involved, and must almost inevitably lead to infection of the general peritoneal cavity. The only

ACUTE PHLEGMONOUS GASTRITIS

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treatment which offers any prospect of success is gastrectomy, and that only in cases where the process is localized and the line of section can be made through approximately normal stomach. Where the disease is secondary to cancer of the stomach even total gastrectomy is likely to be futile, and where the whole stomach is involved any form of operative treatment seems useless.

REPORT OF CASE

On Sunday night, Aug 21, 1921, a stout woman, age 60, who had previously been in good health, was suddenly seized with intense abdominal pain and vomiting. Dr F. Callaghan saw her late on the following night (Monday) and sent her immediately to hospital, where I saw her in consultation with him early on Tuesday morning.

EXAMINATION—Her temperature, which had been 104° the previous night, was now 102.5° , the pulse-rate was 92, and the respirations 26. She looked gravely ill, and was groaning with pain, which she referred entirely to the epigastrium. She had vomited since admission, but there was nothing abnormal about the vomit. The tongue was dry and brown, respiration somewhat laboured, and she complained of great thirst. There was dullness at the base of the left lung, and a mitral systolic murmur. Examination of the abdomen showed distinct rigidity of the epigastrium, which was tender on deep palpation, the tenderness extending to the left of the middle line. The rest of the abdomen moved with respiration, was flaccid, and not tender. The gall bladder was not palpable, and there was no alteration of liver dullness. Rectal examination was negative. Urine negative. Questions about her previous health elicited only a vague history of dyspepsia extending over years, for which she had never sought medical advice.

The diagnosis was a matter of great difficulty, and the following possibilities were considered:

1 *Gangrenous Cholecystitis*—This was suggested by the sudden onset, high temperature and evidence of severe septic absorption, but negatived by the absence of tenderness or rigidity in the gall bladder region, while the gall bladder was not palpable.

2 *Perforated Gastric Ulcer*—Was favoured by the acute epigastric pain, tenderness, and rigidity, but the high temperature and absence of signs of general peritoneal involvement at this stage were distinctly against such a view.

3 *Acute Pancreatitis*—Could be neither affirmed nor excluded.

In the absence of a definite diagnosis it was nevertheless felt that we were confronted with an urgent inflammatory condition in the upper abdomen which demanded exploration. Immediate operation was advised.

ANESTHESIA—Gas and ether was administered by Dr. Callaghan.

OPERATION—Assisted by Mr. H. MacAnlevy, I made a right pararectal incision. There was no extravasation, no free fluid, no fat necrosis, and no signs of peritonitis. Gall bladder, appendix, liver, and pancreas were all normal. The upper reaches of the stomach were very peculiar—it was hyperemic and adenomatous, its walls so thick that one could hardly palpate a lumen. It felt heavy, boggy, and melastic, and could be brought up for inspection only with great difficulty. The whole upper meses were those of acute inflammation of the stomach, which was somewhat thicker than the rest of the organ, a small amount of clear fluid slightly blood stained was noticed in Morrison's pouch—apparently exudate from the inflamed stomach. The process involved the whole stomach, the interior wall of which showed numerous subperitoneal hemorrhages, especially along the greater and lesser curvatures and at the cardiac end. There was no evidence of a perforation of either anterior or posterior wall. Owing to the fatty condition of the omentum it was difficult to detect glands, but I fancied I could palpate an enlarged gland in the gastroduodenal omentum towards the pyloric end. There were no perigastric adhesions.

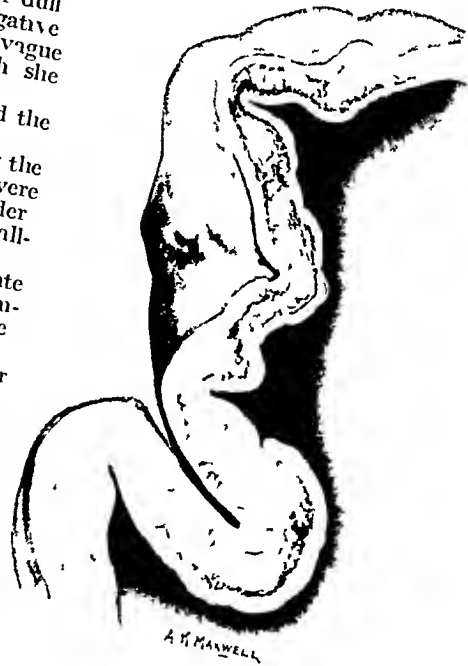


FIG. 1.—Section of stomach wall from the cardia to the pylorus, showing thickening and suppuration.

While examining the pyloric end, I found a small amount of clear fluid slightly exudate from the inflamed stomach. The interior wall of which showed numerous subperitoneal hemorrhages, especially along the greater and lesser curvatures and at the cardiac end. There was no evidence of a perforation of either anterior or posterior wall. Owing to the fatty condition of the omentum it was difficult to detect glands, but I fancied I could palpate an enlarged gland in the gastroduodenal omentum towards the pyloric end. There were no perigastric adhesions.

From the appearances of the stomach, I judged the condition to be acute phlegmonous gastritis. The only treatment that suggested itself was to make incisions into the œdematous walls, but as the entire stomach and both surfaces were obviously involved, I felt that any treatment would have been futile. The abdomen was therefore closed and the patient returned to bed. Death took place four days from the onset of symptoms. Marked abdominal distention indicated a terminal peritonitis.

No complete autopsy was possible—but the wound was re-opened after death. The peritoneal cavity was full of dirty grey exudate. The stomach removed showed a tiny perforation in the midst of an ecchymotic area on the interior surface near the cardia. On splitting the organ along the greater curvature, the walls were seen to be unusually thick (2 in in places), with a layer of pus in the submucosa extending from cardia to pylorus. Pus exuded freely on compressing the edges, and when incisions were made into the posterior wall for the purpose of obtaining cultures, fluid pus poured out.

Bacteriological examination of the pus revealed a pure culture of *Streptococcus brevis*. The appearance of the cut edges is shown in the excellent coloured drawing made for me by Mr A K Maxwell (Fig 51). Careful examination of the mucosa failed to show any lesion.

MICROSCOPIC APPEARANCES.—Sections were cut from the thick pyloric and less thick cardiac ends. In both the submucosa is greatly thickened and shows many small round cells, especially polymorphonuclears. Actual œdema is apparent. In sections stained by the Gram Weigert method many cocci are seen, often arranged in short chains. The intact and excellent condition of the mucous membrane is noteworthy.

I am greatly indebted to Dr E W Bowell, of the Clinical Research Association, who very kindly examined the slides. He could find no evidence of neoplasm in any of the sections, and regarded the condition as purely inflammatory.

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ACUTE PHLEGMONOUS GASTRITIS

ILLUSTRATION AND NOTES OF A CASE FURNISHED BY SIR BERKELEY MOYNIHAN

The patient, a boy, age 17, was taken ill a few hours after eating pork pie. He complained of acute abdominal pain, and there was exquisite epigastric tenderness.

The epigastrium was distended, and the abdomen, inflated in its upper part but retracted below, presented a very striking and unusual appearance.

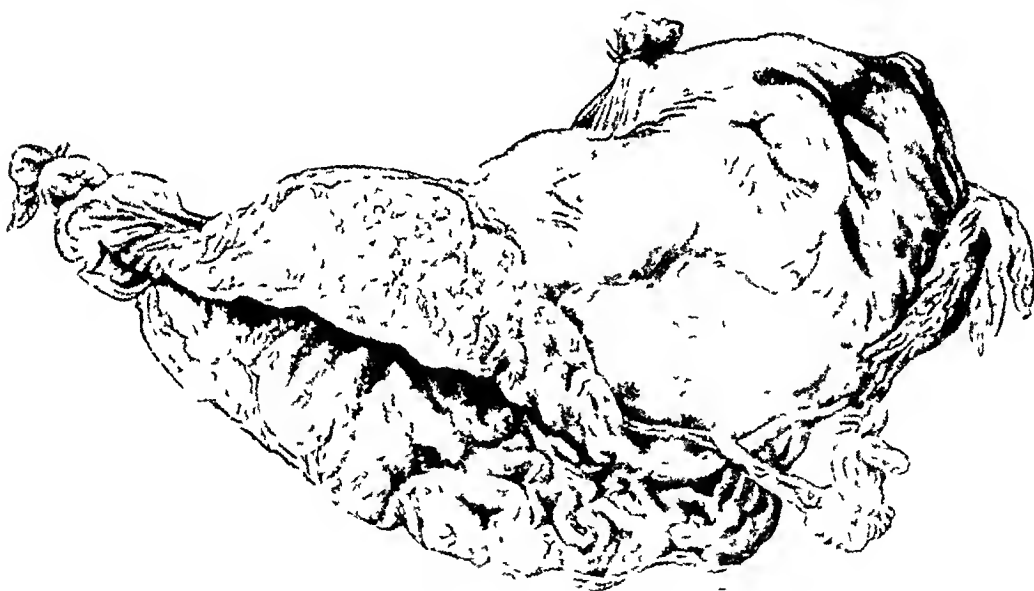


FIG 52.—Sir Berkeley Moynihan's case of phlegmonous gastritis.

The boy was very ill, the pulse was never less than 116, there were collapse, vomiting, and profound prostration, which ended in death about thirty-eight hours after the onset of symptoms. At the post mortem a typical and most acute phlegmonous gastritis was found. No lesion of the mucous membrane of the stomach could be seen (*Fig 52*).

TUBERCULOUS CHANCRE

By J. A. NIXON AND A. RENDLE SHORT, BRISTOL

THERE is a variety of tuberculosis of the skin which so closely resembles a primary syphilitic sore that, on the rare occasions when it occurs it is likely to be diagnosed as extra-genital chancre. Hitherto, if this lesion has been recognized at all, writers have included it under the term tuberculosis verrucosa cutis, or verruca necrogenica. It is true some authors mention that *T. verrucosa* has occasionally to be distinguished from extra-genital chancre, but they fail to see that when a tuberculous ulcer resembles a syphilitic chancre it has not the wart-like appearance that justifies the term 'verruca'. Thus it happens that the diagnosis of verruca necrogenica does not suggest itself to the observer, and the possibility of the lesion being tuberculous is overlooked.

Gaucher and Hutinel¹ give instances of inoculation tuberculosis, including Tseherning's frequently quoted case of a servant girl's finger inoculated by the broken spit-cup of a consumptive. Sequeira,² in his description of *T. verrucosa*, says that two types may be recognized. In the first a small red swelling develops at the site of inoculation, and upon it a small pustule appears. "The swelling slowly enlarges to form a warty nodule with an infiltrated base, surrounded by a zone of erythema. . . . the lymphatic glands enlarge early. In one such case where the lesion was at the root of the nose, we were for some time in doubt whether the sore was not syphilitic, as there was a hard bubo under the chin." The same author in *Allbutt and Rolleston*³ speaks of some cases of *T. verrucosa* that have to be distinguished from blastomycosis, extra-genital chancre, and carcinoma. Gaucher⁴ mentions tuberculous ulcers of the skin, which, he adds, should not be mistaken for hard chancre, since the latter has an infiltrated base, no tendency to extend, and is accompanied by glandular induration—characters that he evidently does not ascribe to the tuberculous lesion. Wilson,⁵ describing a series of cases of primary tuberculosis of the penis following circumcision, says that the condition has been mistaken for syphilis, chancreoid, and cancer.

But the majority of these descriptions either specifically state that the lesion is wart-like, or make use of the term 'verruca'. In the cases dealt with in this paper none of the lesions was in any degree wart-like, and so long as inoculation tuberculosis is described as causing a warty or verrucous lesion, observers who rely on book descriptions will fail to diagnose the true nature of the cases.

In certain individuals direct implantation of tubercle bacilli into the skin by means of a cut or abrasion gives rise to a localized indurated papule. This papule develops into a small indolent ulcer of cartilaginous consistency, having an edge that is slightly ramparted and translucent. It is attended by enlargement of the nearest group of lymphatic glands, which may be mistaken for a sentinel bubo. The induration of the ulcer causes it to be mistaken for extra-genital chancre, although syphilitic chancres of the skin are usually not indurated but assume a raspberry appearance which the tuberculous ulcer never possesses. Sometimes the tuberculous ulcer may look exceedingly like rodent ulcer, but in the latter case there is an absence of glandular enlargement.

These primary tuberculous ulcers, which from their appearance and behaviour we have called tuberculous chancres are probably due to inoculation of tubercle bacilli into persons who have a latent tuberculous infection. Their tissues become intolerant to the presence of the bacilli. The ulcer at the site of a subsequent inoculation represents the efforts of the tissues to expel the bacilli by local necrosis where they have lodged, Koch first described this peculiar reaction which results from a previous infection, and it is known as Koch's phenomenon.

The diagnosis of tuberculous chancre can only be made by excising part or the whole of the ulcer and examining the tissue for tubercle bacilli, either by staining or, if this proves negative, by animal inoculation. A negative result should never be accepted without animal inoculation.

The following cases illustrate the character and behaviour of this form of tuberculous lesion —

Case 1—*Mrs B*, age 57, sent by *Dr Myles*, of *Clifton*, came under observation in *October, 1920*, complaining of a pimple on her chin for the past four months. She thought that it had been caused by her son kissing her. He was dying of pulmonary consumption.

On the point of her chin there was a raised papule about one centimetre in diameter. It was pale pink in colour, not ulcerated, but had a slightly ramparted edge, and felt as hard as a true Hunterian chancre; it was non-adherent to the deep structures. Below it there were a few solitary transparent nodules in the skin, the size of pin's heads. The submental gland was enlarged to the size of a walnut and constituted a sentinel bubo.

The sore and glands were excised. Microscopic sections showed the presence of tubercle bacilli, and animal inoculation (undertaken by *Professor Lyle Cummins*) demonstrated that the bacilli were of human type. The skin of the first operation broke down and a second wider excision led to complete and firm healing. The sections are shown in *Figs 53, 54*.



FIG. 53—*Case 1*. Section of tuberculous chancre (low power).

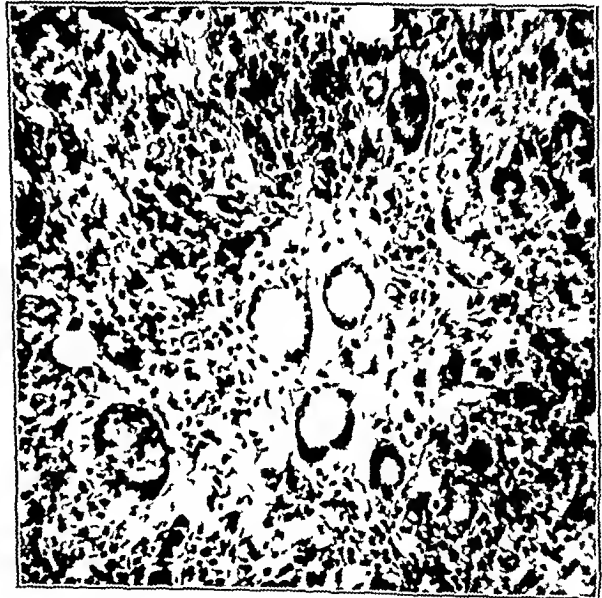


FIG. 54—*Case 1*. Section of tuberculous chancre (high power).

Case 2—*Margaret N*, age 6, fell and cut her knee while at the seaside in *May, 1919*. A small ulcer formed which remained open for three months. The ulcer was as large as a florin, situated over the middle of the right patella. Its colour was pale purple. The centre was raw, and discharged only scanty serum, showing no sign of granulation. The margin was raised, and formed a rolled-in rim, not undermined. The ulcer was indurated and non-adherent to the patella. A large bubo formed in the groin which suppurated.

In *August 1919* the ulcer was excised and a large mass of glands dissected out of the groin by *Mr Burton* of *Cromer*. The child made an uneventful recovery and shows no other sign of tuberculosis. Tubercle bacilli were demonstrated in the skin of the ulcer and in the glands. There was no evidence of tuberculosis in any member of the family or of the household.

Case 3—*Edw. P*, age 13, was admitted to *St Bartholomew's Hospital* in *June 1900*. In *April* of that year he had fallen and struck the left side of his face on a desk at school. The left eye was bloodshot next morning. Three days after the accident a painful swelling developed in front of the left ear. This suppurated and was cured a month later. During the whole time the conjunctiva of the left eye had remained inflamed. A small hard tumour was found on the conjunctiva in the form of a nodule at the external canthus, with swelling of the lids and general injection. The surface of the lump was ragged, tuberculous, and ulcerated at its lower part, covered with a thin puriform secretion. The rest of the inner surface of the lower lid was studded with small translucent granules not unlike milium tubercles. One was situated on the corneal margin, looking like a phlyctenule. The pre-auricular gland on this side was swollen and still discharging pus.

Mr Jessop diagnosed the case as a syphilitic chancre of the eyelid. Mr Alfred Willett and Mr Vernon thought it was probably tuberculous. The tumour and the gland were excised by Mr Jessop, and proved by animal inoculation (Professor Andrewes) to be tuberculous. The boy made an excellent recovery.

Case 4—A B, age 34, widow living in the country. Patient was seen in November, 1921. She complained of a nodule on the upper lip, with a two months' history. It began as a small vesicle in the middle line, on the mucocutaneous margin, a week after a child bumped up against it and bruised it. Recently it had been growing rapidly.

On examination there was found a red proliferating growth, about the size of a filbert, extending from the filtrum of the nose to the border of the lip, and slightly overhanging the lower lip (*Fig. 55*). It was firm, slightly ulcerated on the surface, and redder than a primary



FIG. 55.—*Case 4*. Tuberculous chancre of the lip. Shows the dusky red hypertrophic swelling, the superficial ulceration, and the enlarged gland in the neck.

chancre usually is. There was a hard indolent lymphatic gland beneath the right lower jaw in the submaxillary region. There were no secondaries. Wassermann was negative. Temperature normal. It was regarded as without doubt a primary chancre with the usual bubo. Four professional surgeons saw it, and all thought it typical. Several of them had acted or were still acting as venereal clinic special officers.

Four doses of novarsenobenzol were given, but the condition did not improve, beyond healing of the superficial ulceration. It was therefore excised, and the pathological report showed unmistakable tuberculosis. She did well.

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PLASTIC REPAIR OF THE FACE AND HAND.

By J J M SHAW, EDINBURGH

As exemplifying two of the methods which have proved of great service in plastic repair—the tube pedicle, and the Thiersch graft on mould—the following description of the disablements and treatment of a severely-burned patient may be of interest. In a recent article by Lieut-Colonel H P Pickernill and Mr J Renfrew White, the application of the tube-pedicle method to gunshot wounds of the face and to areas of chronic ulceration in the limbs was set forth with great clearness (*British Journal of Surgery* January, 1922, p 321). The present paper will therefore be restricted to the treatment of the effects of burns in an illustrative case.

L R B, age 20, of the R A F, was burned by ignition of petrol in a motor launch in Poole harbour in September, 1919. All his shipmates succumbed, and for a considerable time the patient's own chance of survival was uncertain. He was treated for more than six months in a local hospital, in which efforts were directed mainly to saving his life—the prevention of his contracture deformities, in the early months at least, being relegated to a secondary position. Later he was sent to Woolwich Military Hospital, and thence transferred to my wards at Queen's Hospital, Sidcup, in March, 1920.

The illustrations of the progress of the case are almost self-explanatory, but a brief description of the operations will make the various steps more clear.

The Face (Figs 56-62)

1 A strip of skin and platysma, 5 in long by $1\frac{1}{2}$ in wide, was dissected up on each side of the neck. The upper ends were left attached, and the lower remained united by a bridge formed by a portion of the skin and subcutaneous tissue of the chest which corresponded exactly to the requirements of covering for the chin as measured by a pattern of tinfoil. The neck strips were then tubed by suture of their free margins with vaselined linen thread, and the skin edges bordering the raw areas were undercut and sutured together beneath the pedicle with silkworm gut. In this first stage, the bridge was demarcated, but only partially freed, in order to avoid too great a primary demand upon the circulatory supply and lymph escape via the pedicles. The raw areas in the neighbourhood of the bridge were dressed with ambrine. The areas of skin supply are shown in the diagrams.

2 A month later, the dense scar-tissue of the chin was removed. The bridge, having been completely severed from the chest, was turned up into position and sutured with three points of catgut, one at the symphysis and one at each foramen menti, in order to create slight natural depressions, and with horsehair around the lip margins and lower border of the chin, where comparatively healthy skin adjoined. A group of tiny epithelial plants from the abdomen were spread over the raw surface of the chest, according to the Ollier-Thiersch technique, and dressed with gauze wrung out of normal saline. This area rapidly epithelialized, and, with massage and oilunction, the scar ultimately became soft and pliant.

3 After the lapse of ten weeks, such scarred skin of the nose as remained was turned down, as shown in the diagram, to form a nasal lining. The neck ends of the pedicles were severed, partially opened out, and turned up symmetrically to form the nose.

4 Three weeks later, the unhealthy tissue was removed from the upper lip. The pedicles were divided at the tip of the nose and sutured into the raw area, the free ends meeting at the mid-line of the lip.

Thus in four operations the new coverings for chin, nose, and upper and lower lips were provided. No dressings were used upon the face at any stage. The extent of



FIGS 56-61—From photograph illustrating the progress of the operations on the face

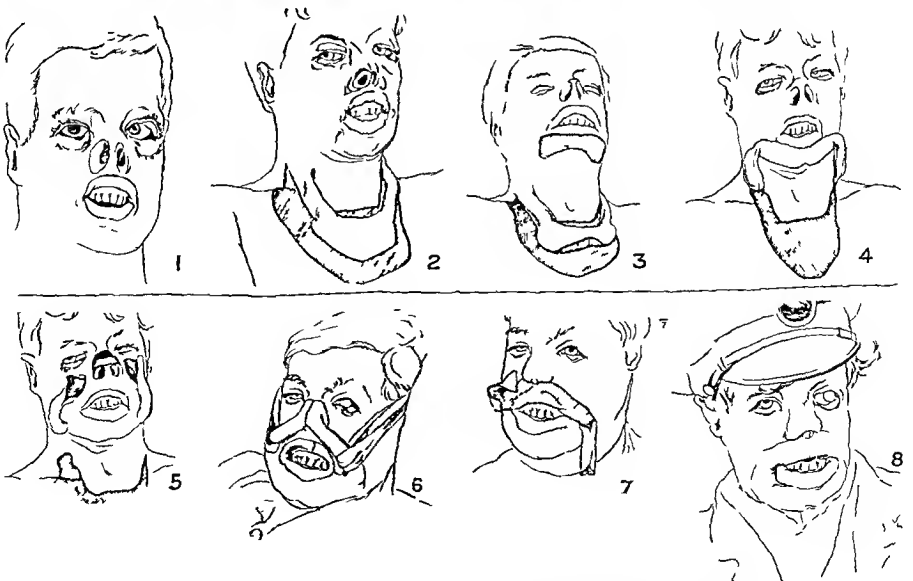


FIG C2—Diagrams showing stage (1-8) of the operations on the face

retraction at the root of the nose and in the new lining of the nostrils was slightly underestimated, with the result that the damaged columella was incompletely concealed, but a small prop of cartilage or local advancement at a later stage will easily remedy this defect. Freed from attachment in the circumoral region, the remaining portion of the skin of the cheek retracted satisfactorily and pulled the scar upwards and outwards at the line of union to the position of the normal nasolabial fold. It also softened to a remarkable degree, and lost its florid and rough look. The final result of this relaxation was a weather-beaten appearance over and below the malar prominence where the colour is usually somewhat deepened. The treatment of the ectropion of the lower lids also helped towards this end.

The Eyes (Figs 63-65)

The dressings shown in the early photograph (*Fig 63*) were necessitated by the



FIGS 63, 64—The eye of the patient open and closed before and after the operation

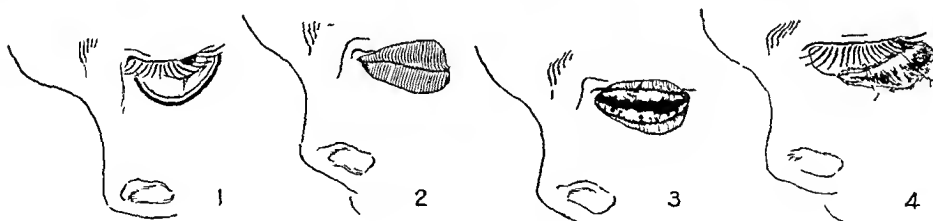


FIG 65—Diagram of the steps of the operation (1-4)

condition of the scalp, and by the ulceration over both temporomandibular joints when admitted.

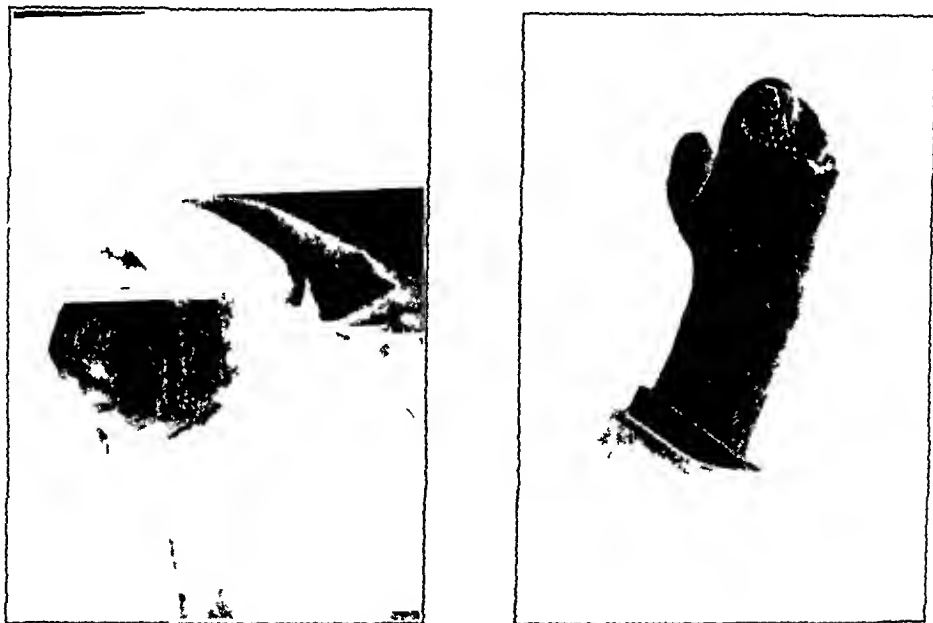
The ectropion of the eyelids, both upper and lower, on both sides, was dealt with by the application of Thiersch grafts upon moulds. This method has been fully described in his admirable book by my late senior colleague, Mr H D Gilhes, by whom its utility was early recognized and applied in inlays of the mouth, nose, and eye sockets, and for ectropion.

The dental composition known as 'stent' is softened by heat and then pressed into a hollow created by an incision alongside the ciliary margin and the freeing of the lid by dissection. The mould, when set, is removed, and around it is wrapped a Thiersch graft raw surface outwards. Graft and mould are re-inserted into the prepared bed and almost buried by cross-suturing. Ten days later, the mould is removed and the cavity is found to be lined with smooth and healthy epithelium. This process is usefully practised in

the ectropion following severe lupus, indeed, the whole clinical picture of this case was not dissimilar to the effects of that disease when it has been allowed to progress beyond the stage of primary excision followed by plastic restitution, which, as for rodent ulcer appears to be the most reasonable line of treatment in those cases in which local applications have been thoroughly tried but have proved ineffective. The accompanying diagrams (*Fig 65*) serve to illustrate the method, and the photographs, with eyes closed and open before and after operation, show the degree of restitution which was effected.

The Hands (*Figs 66-68*)

As the photograph of the right hand indicates the hands and wrists were very



FIGS 66, 67—Hand before and after operation

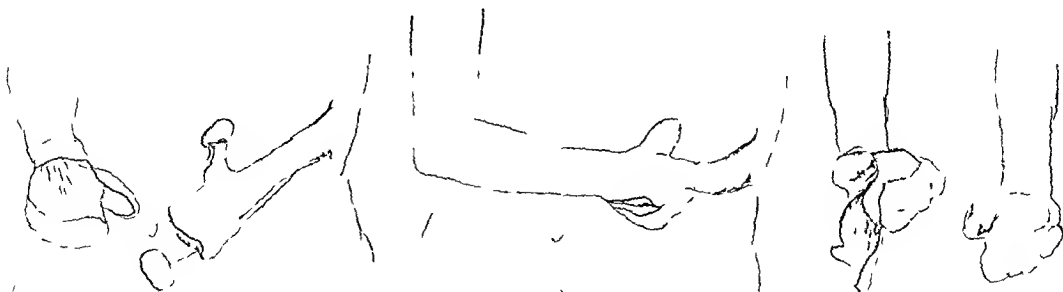


FIG 68—Showing four stages of the grafting operations

severely burned. The wrists were fixed in a position of extreme flexion and large unhealed areas were present on the dorsal aspects, which had been dressed for many months with the accompaniment of severe pain. The palms were filled with thickened and sodden masses of epithelium. On the left side two sinuses led down in the direction of the head of the middle metacarpal bone. The distal phalanges of all fingers had disappeared, and several necrosing pieces of bone protruded from the shapeless masses which represented the fused remains of fingers and thumbs of both hands. Small blebs on the dorsal aspects, which occasionally broke down and emitted foul-smelling sebaceous matter,

indicated the sites of the digital clefts. On account of the fusion of the stumps in dense scar-tissue, no trace of independent movement was discernible in fingers or thumbs, although the patient still preserved a subjective sense of control of each digit. Radiographic examination indicated, and subsequent dissection confirmed the fact, that actual synostosis had taken place between the proximal phalanges of the left middle and ring fingers.

The patient was right-handed. Sepsis and pain were greater on the left side. It was impossible to obtain from the abdomen sufficient covering for both hands without the production of a disabling scar, as, for the right hand alone, I estimated that 38 square inches of skin were required. In view of these facts, amputation was performed at the lowest level of unscarred skin in the middle third of the left forearm, and an artificial arm of the 'Cauet' type, fitted later, gave him a limb of considerable usefulness.

1 For the right hand, an abdominal pedicle was cut, 9 in long by 3 in wide slightly aslant the natural skin fold, and in the vascular and trophic line of supply. At right angles to the main pedicle, a thumb extension was also fashioned. By a little undercutting of the skin and relaxation by flexion at the hips, the edges were easily apposed beneath the pedicle, and primary union resulted.

2 The second stage was carried out three months later, but this long interval was due to the difficulty of so reducing the sepsis of the hand that no unnecessary risk of losing a portion of the pedicle by this means would be incurred on attachment.

The dorsum of the hand was cleaned, the extensor tendons defined, and the thumb freed by removal of the dense scar-tissue which had bound it to the stump of the index finger. The medial ends of the pedicle and thumb extension were divided and opened out. The hand was then held in position while the new coverings were sutured over the raw areas. The remainder of the pedicle, designed for ultimate application to the palm, was thus left unopened at this stage, and its range of movement obviated the need for absolute and uncomfortable fixation, thus, with the absence of any raw surface on hand or abdomen, is of considerable importance in view of the discomfort and sepsis occasionally produced by the 'flap' method in this region.

3 In three weeks' time sound union had been established, along with a dependable reverse circulation, which had been accelerated by frequent constriction of the abdominal attachment. The pedicle was severed, opened, and applied to the palm and radial side of the stump of the index. On healing, this produced a full thumb sulcus and a pliable web which gave excellent movement. The patient could write well, and for the first time in a year was able to feed himself with a fork or spoon.

4 A second pedicle was cut on the right side of the abdomen to cover the remainder of the hand, consisting of the ulnar border, the stump of the little finger, a concavity created by the removal of the stumps of the middle and ring fingers, together with the heads of their corresponding metacarpals and, finally, the outer aspect of the thumb. This was carried out in stages of operation similar to the other.

The operations for facial repair, for the eyes, and for the hands, were carried out concurrently, and necessitated fourteen general anaesthetics of gas and oxygen, which were administered intratracheally except when treatment was directed only to the hands or abdomen.

The patient's health improved steadily as his septic foci were eliminated and as he was enabled to go about in the open air with eyes adequately protected from dust and glare, and with the consciousness that his appearance had ceased to be repulsive. The psychic effect of any obvious disfigurement, whether due to trauma, disease, or a congenital affection such as nevus, is always a factor worthy of consideration, and it was pleasing to note the steady mental uplift in this stout-hearted lad as his many disablements were ameliorated.

A CONTRIBUTION TO THE PATHOLOGY AND ETIOLOGY OF OSTEO-ARTHRITIS: WITH OBSERVATIONS UPON THE PRINCIPLES UNDERLYING ITS SURGICAL TREATMENT*

By A. G. TIMBRIFIL FISHER, LONDON

INTRODUCTORY

THERE can be no doubt that the condition called by English writers 'osteo arthritis' has afflicted not only mankind but the lower animals, from remote periods in the earth's history. We find its stigmata in the skeletons of prehistoric animals and in human remains of considerable antiquity, for among the bones of ancient Egyptians at the Royal College of Surgeons of England are several exhibiting these changes, and in hieroglyphic writing the 'determinative' for old age was the figure of a man crippled with arthritis. Moreover, we find no race or clime to be exempt from the disease.

The pathological changes are of very great interest and importance from their extraordinary diversity, for in the same joint we may see the phenomena of repair, inflammation, and new growth merging indistinguishably into one another. Moreover accompanying or following the cellular proliferations, are unequivocal signs of degeneration. A striking feature, and one which distinguishes the disease from the more acute and probably infective group of conditions known as 'rheumatoid arthritis', is the almost invariable absence of small-cell infiltration.

There can be little doubt that in osteo-arthritis we are faced with a borderland between a frankly inflammatory condition and a neoplasm of the joint structures.

The etiology of osteo arthritis presents us with a difficult problem. It would appear, however, that research into this subject is urgently needed, not only in order that we may be able, from a knowledge of the cause, to treat this extremely common, universal, and crippling disease on scientific lines, but, in addition, such a line of research may eventually throw some light upon the pathogenesis of neoplasms.

In this paper a section is devoted to some largely experimental observations upon joint physiology, because it was felt that our knowledge concerning the basic principles of the physiology of the joints leaves much to be desired, and it was thought that a study of this might throw some light upon the peculiar pathological features of osteo arthritis. The grosser anatomical features of the disease have been so carefully described by Adams and others that a description of these is purposely omitted. The morbid histology of the earlier stages, which appears hitherto to have received less attention than it deserves, has been investigated more fully in order to ascertain what light these earlier changes might throw upon etiological problems. Symptomatology and treatment are given in a summarized form from considerations of space, and will receive more detailed notice elsewhere. The etiological observations are of a preliminary nature, as research is being continued into this aspect of the problem.

Nomenclature—Much of the existing confusion concerning osteo arthritis can be traced to the fact that many writers have invented descriptive terms for the disease in which an attempt is made to embody what appears to be its principal pathological characteristics. We have already noted how manifold and diverse these changes may

* Embodying the Hunterian lecture delivered at the Royal College of Surgeons of England and constituting an abstract of a preliminary report to the Medical Research Council who have generously defrayed the expenses of the research.

be This appears to be the explanation of the fact that certain American workers have christened the disease by names which indicate diametrically opposite conditions

Goldthwait, for example, classifies chronic arthritis into (a) Infectious arthritis (b) Atrophic arthritis, (c) Hypertrophic arthritis Nichols and Richardson have introduced the terms 'proliferative' and 'degenerative' arthritis, corresponding respectively to the 'atrophic' and 'hypertrophic' arthritis of Goldthwait, or to the 'rheumatoid arthritis' and 'osteo-arthritis' of English authors

EXPERIMENTAL AND OTHER OBSERVATIONS UPON THE APPLIED ANATOMY AND PHYSIOLOGY OF ARTICULAR CARTILAGE

WITH SPECIAL REFERENCE TO ITS STRUCTURE MODE OF NUTRITION AND TO THE REPAIR OF WOUNDS THEREIN

It is a healthy sign that modern physiologists are devoting attention to the structure and physiology of the individual cells—a movement that must lead ere long to significant revelations In pathology it is only by a study of the individual cells under abnormal conditions that we can hope for a clearer comprehension of disease

The complicated problems connected with the etiology and pathology of osteo-arthritis are so intimately bound up with the structure and physiology of the articular cartilage that it is essential to have a clear idea of this structure and physiology My researches into the latter furnish, I venture to believe, the clue to many of the pathological phenomena of osteo-arthritis

Let us before discussing the anatomy and microscopic structure of articular cartilage, glance at another specialized form of hyaline cartilage

Costal Cartilage—The periphery is formed by a connective-tissue perichondrium As these connective-tissue cells are traced towards the deeper parts the cells become larger, although still of connective-tissue type, and not surrounded by any matrix If traced still further, these cells indistinguishably merge with the proper cartilage cells The latter, in the central portion of the cartilage, are seen to be large, somewhat angular cells occurring in groups of two three, or four In sections of adult costal cartilage stained by carbol-thionin, Professor Shattock observed that the matrix immediately surrounding the cartilage cells stains a deep claret colour (*see Fig 72*) This more deeply stained portion of the matrix is evidently of more recent origin and contains a larger proportion of muem to collagen than the older matrix, since, as is well known, carbol-thionin stains muem freely There can be little doubt that the central groups of cartilage cells are derived from the peripheral by a process of proliferation Occasionally, ill-defined fibres can be seen in the matrix of costal cartilage, and, as age advances, lime salts may be deposited in the matrix, or as is well known, the cartilage may become completely ensheathed with bone continuous with the rib and sternum

Repair in Costal Cartilage—It appears that the fully formed cartilage cells in the centre have little power of undergoing proliferation, and that repair takes place by means of the perichondrium

Experiment 1—A median longitudinal section was made in the 5th costal cartilage of a rabbit through its whole thickness Ten weeks later microscopic examination of the divided portion of costal cartilage reveals that the cartilage cells on either side of the incision show no sign of proliferation but that there is a thin strand of connective tissue derived from the perichondrium occupying the cleft in the centre of the costal cartilage

The Vitality of the Cartilage Cells—In a Hunterian lecture upon loose bodies in joints, I demonstrated that in those loose bodies of the traumatic or 'classical' type which contain both cartilage and bone, and which have been for some time quite free in the joint cavity, the cartilage cells retain their vitality, whereas the majority of the bone cells die To test further the behaviour of cartilage cells when transplanted beneath the skin of their host, I would adduce the following experiment —

Experiment 2—A portion of costal cartilage was resected with its perichondrium and placed in saline solution at body temperature. A separate incision was next made through the skin covering the anterior aspect of the left side of the chest, and the portion of costal cartilage was introduced through this and pushed up towards the axilla. The small incision was separately sutured. Ten weeks later the portion of costal cartilage was found to be firmly incorporated with the subcutaneous tissues, and was removed.

Microscopical examination shows the cartilage cells to have retained their normal characteristics and to contain well stained nuclei. At the periphery the perichondrium can be seen, and external to this a delicate investment of connective tissue derived from the subcutaneous tissues.

The marked vitality of the cartilage cell when compared with the bone cell requires emphasis in order to explain many of the phenomena of osteo-arthritis.

Articular Cartilage—A reference to *Fig 69* will show the main characteristics of articular cartilage as it illustrates the different characteristics of the lateral and central parts, to which I hold many of the most striking pathological features are due.

It will perhaps avoid confusion if we discuss the structure of articular cartilage under two headings, viz., the central and the lateral articular area.

1 CENTRAL ARTICULAR AREA—The superficial stratum is seen to consist of flattened cells arranged in groups which lie parallel to the surface. If the immediate surface is carefully examined it will be seen that it is constituted by a well-defined regular curvi-

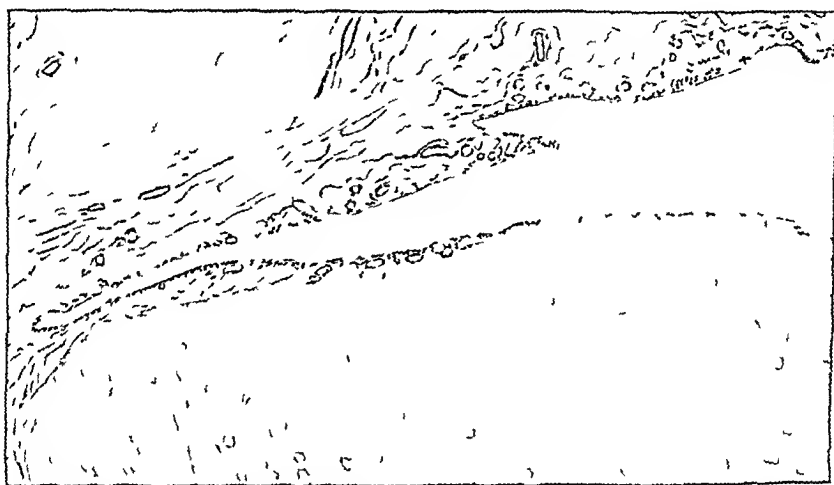


FIG. 69.—A critical section of normal articular cartilage from lower part of human patella showing synovial membrane above which extends for a certain distance at the margin over the articular cartilage. (Two inch obj.)

linear margin which is devoid of any actual cell covering, and is evidently formed of matrix. Henle has described a delicate layer of cells (perichondrium) upon the actual free surface, but investigation convinces me that this must be very rare in the normal adult, although it unquestionably exists at an earlier stage of development. A point of importance is that the horizontal group of cells of the superficial stratum shows no sign of degeneracy or diminished vitality, but even in the case of the most superficial the nuclei stain well, and there is no indication that they are other than normal and healthy cells.

In the intermediate zone the groups of cells are more irregularly disposed, and in the deeper zone the cell groups are arranged vertically. Ogston's main conclusions were that articular cartilage is continually renewing itself from the focus of central growth, that it grows in two directions, and that it develops in the direction of the joint an effete layer that is worn away by the joint movements.

However, examination of normal cartilage reveals no justification for the assumption that it develops towards the joint this effete layer, or that this contributes in any way to the synovial fluid.

It seems clear that, far from being degenerate, these superficial cells are the source from which the deeper cells are derived, and that from developmental and other reasons they are analogous to the superficial cells of the lateral portion of the articular cartilage, or to the perichondrial cells of costal cartilage.

2 LATERAL ARTICULAR AREA (see Fig 69)—The lateral portion somewhat closely resembles those varieties of hyaline cartilage which are furnished with a perichondrium for the surface of the cartilage is covered laterally by a delicate extension of the synovial membrane. When traced centralwards the connective tissue is reduced to a single layer of close-set endothelial cells, these subsequently produce hyaline matrix in which they become buried, so that the more central parts of the investing cartilage consist at the free surface of matrix, i.e. cartilaginous. At the edge the articular cartilage becomes markedly fibrillated, and merges into the fibrous tissue, beyond which it is furnished with capillaries derived from the circulus, without there being any marked increase of cellularity accompanying the transition. In the thin synovial layer over the lateral articulating area lie capillaries derived from the circulus articularis vasculosus of William Hunter. This lateral part is therefore far better nourished than the central. This fact is of fundamental importance and, in my opinion, is the key to many of the phenomena of osteo arthritis, for my theory, as will be seen later, is that the central part of the articular cartilage responds to the cause of osteo-arthritis by degeneration, whereas the lateral part proliferates, owing to its richer nutrient supply.

Development of Articular Cartilage

—This difference in structure of the central and lateral portions of the articular cartilage is capable of an explanation on developmental grounds.

Towards the termination of the second month the joint cavities have appeared—a split occurring in the mesenchymatous tissue, which usually commences laterally. This is well shown in Fig 70, (kindly drawn for me by Dr Gladstone).

At the fourth month of intra-uterine life the surface of the joint cartilage is still covered by this layer of connective tissue. This is the stage which is normally found throughout life in certain birds—notably the ostrich.

With the increasing movements of the child before birth the perichondrial layer gradually recedes from the more central parts of the articular surface, but at birth it still strays for a little way over its edge (Fig 71). Microscopically, however, the connective-tissue layer can be traced beyond the naked-eye delimitation.

Shortly after the child begins to walk the extension of the synovial membrane over the lateral margin of the articular cartilage makes a further slight regression, but soon assumes its permanent relationship. No perichondrium is present over the more central part, yet there can be no doubt, from a study of their structure, staining reactions, and of their development and comparative anatomy, that these cells have the same function

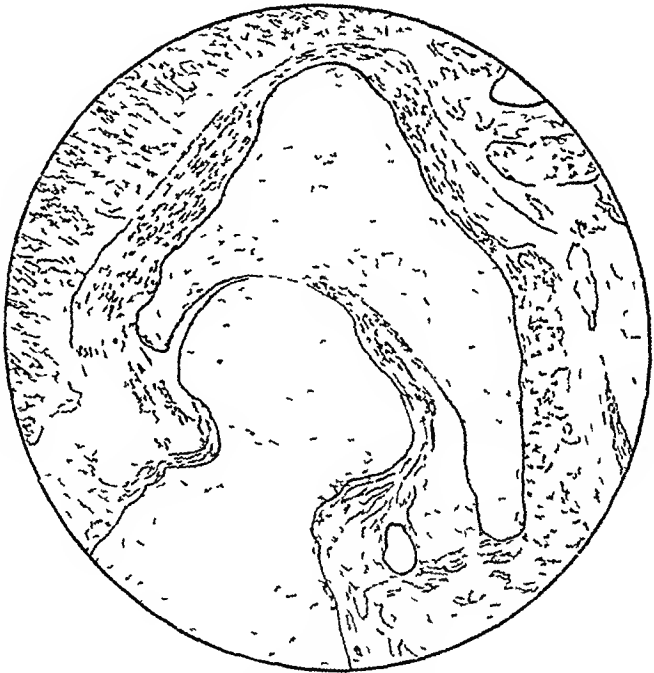


FIG 70—Section through hip joint of human embryo 4.5 cm (9th to 10th week) showing commencement cleavage of the mesenchyme (Drawn by Dr Gladstone)

as the perichondrial cells of the lateral part of the articular cartilage and are the parent cells of the more fully-developed cartilage cells in the deeper parts

Nutrition of Articular Cartilage—This is another question of fundamental importance, and one concerning which there has hitherto been a grave lapse in our knowledge. In a paper entitled "Of the Structure and Diseases of Articular Cartilages" William Hunter first described the *circulus articularis vasculosus* lying near the margin of the articular cartilage, and in younger subjects sending off-shoots on to the articular surface.

He states "The distribution of the blood-vessels to the articulating cartilages is very peculiar, and seems calculated for obviating great inconvenience. Had they run on the outer surface, the pressure and motion of the two cartilages must infallibly have occasioned

frequent obstructions, inflammations, etc., which would soon have rendered our motions painful, and at last entirely deprived us of them. But by creeping round the cartilaginous brim where there is little friction, or under the cartilage, where there is none, they are perfectly well defended from such accidents."

The outstanding fact remains that Hunter believed that the *circulus* plays an important part in the nutrition of articular cartilage.

From the developmental point of view and from that of comparative anatomy, the extent to which the minute branches of the *circulus* penetrate over the articular cartilage depends upon the degree of extension of the synovial membrane over its surface.

Spermen B 171, RCS Museum, shows the *circulus articularis vasculosus* in the knee joint of an ostrich, and was probably injected by William Hunter himself nearly two hundred years ago, and in *Fig 71* is shown the condition in the human full time

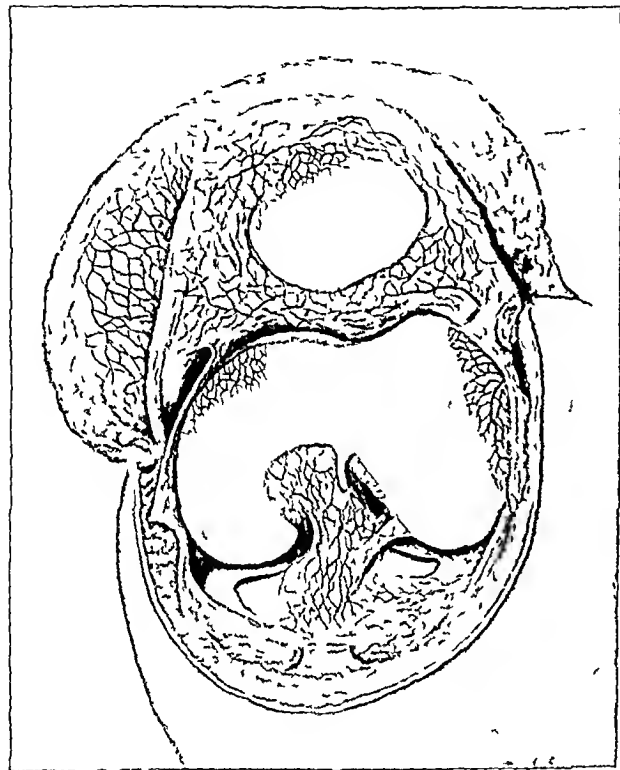


FIG 71.—Blood vessels of synovial membrane and *circulus articularis vasculosus* in injected knee joint of human full time foetus.

foetus. The minute injected vessels stray for a short distance at certain spots over the surface of the articular cartilage. Toynebee came to the conclusion that the principal source of nutrition of adult articular cartilage consisted in the lymph exuded from the large and convoluted vessels lying beneath it in the cancellous spaces.

Recently there has been an attempt to attribute an important part in the nourishment of articular cartilage to the synovial fluid. An argument in favour of this view is the continued growth, while free in the joint, of loose bodies of the 'classical' type. However, a loose body usually acts as an irritant, and causes a greater or less degree of synovitis with an outpouring of fluid rich in albumin which cannot in any sense of the word be designated 'normal' synovial fluid. My analyses, which I now show for the first time, reveal that the normal fluid contains such a low protein content that it is very doubtful whether it plays more than a small part in nourishing the articular cartilage (See Table.)

SYNOVIAL FLUID			TUMOR PLASMA
<i>Average Personal Cases</i>			Human
Total Solids	Human 4.11 per cent	Oven 2.023 per cent	4.2-6.5 per cent
Protein Content	1.6	0.92	1.5-4.3
Mucin	1.95	0.1303	—

My conclusions are as follows —

That the deeper stratum of the articular cartilage is largely nourished in the manner described by Toynebee

That the superficial stratum of the central articular area is nourished by the synovial fluid, and that the remainder of the articular cartilage receives its nutrient supply from the circulus articulari vasculosus. It will be seen that, in the infective and toxic types, this arterial circle is an important medium by which toxins attack the joint.

The subarticular bony lamella cannot be said to form an impenetrable barrier, since after an injection of carmine and gelatin the colour readily permeates this zone. Neither can the calcified zone of the articular cartilage be said to form a serious barrier. The question next arises, How does the nutrient fluid gain access to the cartilage cells? No definite lymphatics with an endothelial lining have ever been demonstrated in articular cartilage. Professor Shattock has recently discovered that if cartilage is stained by carbol-thionin, which stains mucin pink (*Fig 72*), it will be seen that the zone of matrix immediately surrounding the cell groups is stained pink, and is evidently the most recently formed and more mucinous part. In a well stained section it will be seen that the pink zones branch and intercommunicate. In the superficial part of the articular cartilage the meshwork is horizontal, in the middle zone more irregular, and in the deeper zone the meshes lie vertically.

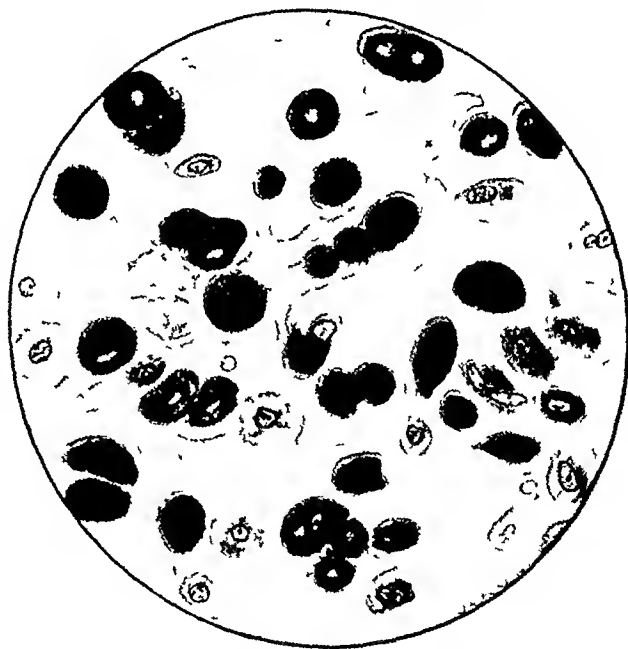


FIG. 72.—Cartilage stained by carbol-thionin which reveals the lines of softer matrix along which possibly the inhibition of nutrient fluid takes place. (Two thirds obj.)

My own theory is that the articular cartilage receives its nourishment from plasma that percolates along the meshwork formed by this more mucinous part of the cartilage matrix which surrounds the cell groups, and the pathological changes seen in the infective and toxic groups of osteoarthritis including the senile strongly suggest that toxic substances percolate along these same nutrient paths.

Repair in Articular Cartilage.—The phenomena of repair in articular cartilage throw considerable light upon osteoarthritis, especially the traumatic type.

Redfern, who performed a large number of experiments upon animals in order to investigate the mode of repair in articular cartilage, came to the conclusion that incisions experimentally made remained open for *long periods* and that when repair took place

eventually, the bond of union was formed of connective tissue derived from the cartilage cells at the margin of the incision. There was no actual formation of new cartilage cells. He also found that incisions in the lateral portions of the articular cartilage healed sooner than those in the central parts. On each side of his incisions there was some feeble and quite local proliferation of the cartilage cells. I have repeated certain of Redfern's experiments, and have performed others which appear not only to confirm the views concerning the nutrition of articular cartilage enunciated above, but also to afford some explanation of the osteophytic developments which are such a marked feature of osteo arthritis.

Experiment 3—Right knee joint of rabbit opened from the inner side. Small portion of articular cartilage with a portion of the subjacent bone chiselled off—the portion involving the inner lip of the trochlear surface of the femur and a portion of the internal femoral condyle. The subsequent changes in this traumatic loose body I have described elsewhere.

The animal was killed twenty-two weeks later, and the articular surface of the femur whence the small fragment had been detached was seen to be smoothly healed over and covered by a white and glistening substance. Microscopical examination revealed the following (Fig 73).

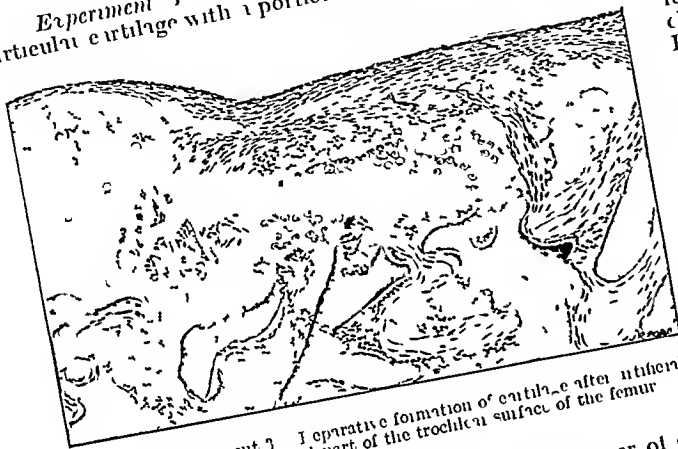


FIG. 73.—Experiment 3. Reparative formation of cartilage after unilateral detachment of the lateral part of the trochlear surface of the femur.

The slight depression in the curvilinear contour represents the spot where the original articular cartilage becomes continuous with the reparative formation over the chiselled surface. The reparative tissue consists of well formed cartilage tissue. Some of this cartilage is probably of the nature of cartilaginous cells, and appears to be derived from the connective tissue cells of the cancellous spaces exposed by the tumour. The more superficial cartilage cells are formed from the connective tissue on the surface, but it is quite clear that much of the cartilage is derived from the deeper layer of the articular cartilage, which can be seen actively proliferating.

This experiment demonstrates that the lateral part of the articular cartilage is capable of a certain amount of repair by formation of new cartilage and this phenomenon can be explained on anatomical and physiological grounds, for we have already shown how the lateral part of the articular cartilage receives far better nourishment than the central parts, and, moreover, is furnished with a perichondrium.

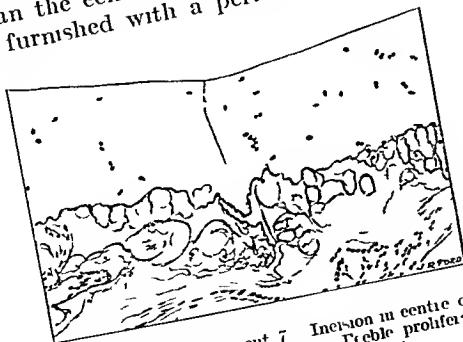


FIG. 74.—Experiment 7. Incision in centre of trochlear surface of the femur. Feeble proliferation of cartilage cells (two thirds obs.).

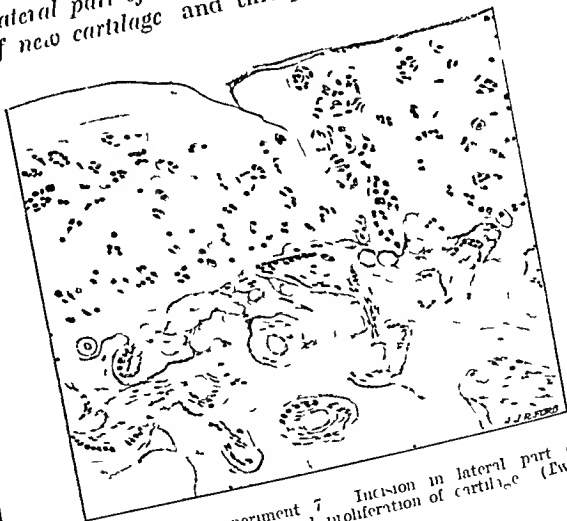


FIG. 75.—Experiment 7. Incision in lateral part of trochlear surface. Marked proliferation of cartilage cells (two thirds obs.).

Experiment 4—Right knee-joint of a rabbit opened and the articular cartilage of the trochlear surface of the femur longitudinally divided, (a) in the centre, (b) near lateral edge. One month later the rabbit was killed and the portion of the trochlear surface examined microscopically. It will be seen from *Figs 74* and *75* that both incisions are plainly visible. There is little, if any proliferation at the sides of the central incision (*Fig 74*), but on either side of the lateral incision (*Fig 75*) there is a well marked proliferation of cartilage cells.

This experiment again demonstrated the greater vitality of the lateral portions of articular cartilage.

EXPERIMENTAL PRODUCTION OF TRAUMATIC OSTEO-ARTHRITIS

Experiment 5—Right knee joint of rabbit. The central part of the articular cartilage covering the trochlear surface of the femur and patellar articular surface was pared away down to the calcified zone, and the joint was closed. After a few days the animal exhibited no limp or any sign of disability. Six weeks later it was killed. The naked eye appearance of the joint is seen in *Fig 76*.

Microscopical examination of a transverse section of the trochlear surface is of considerable interest (*Fig 77*). It will be seen that there is no sign of repair of the pared surface—indeed it has undergone necrosis. The contrast between the necrotic central parts and the lateral portion, which extends for some distance over the central portion, is very marked. The most interesting feature is that the cartilage of



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FIG 76—Experiment 5. Experimental production of traumatic osteoarthritis in knee joint of rabbit ($\times 2$)

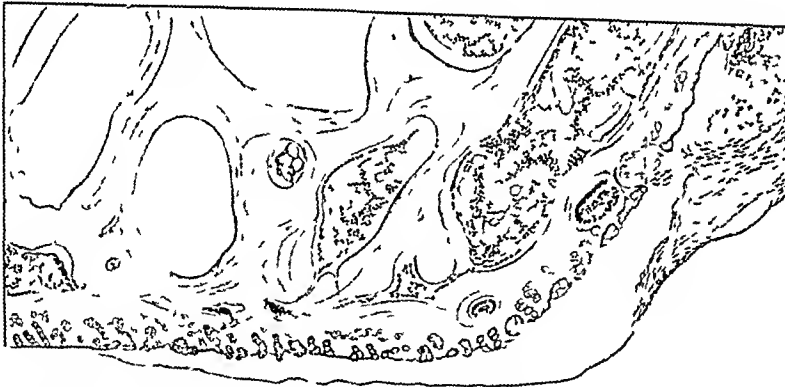


FIG 77 Experiment 5. Compensatory proliferation of lateral part of articular cartilage after experimental removal and damage to central portion. (Two thirds obj.)

the lateral part is in process of active proliferation. The explanation of this occurrence undoubtedly is that the central part, owing to its poorer nutrition, is incapable of repair, and that proliferation of the lateral part must be regarded as compensatory.

Experiment 6—Traumatic osteoarthritis experimentally produced in a rabbit by the action of radium.

A tube containing 0.150 mgrm radium (kindly lent me by Professor Lazarus Barlow) was fixed against the inner border of the internal condyle of the femur near its junction with the trochlear surface. Four weeks later the articular cartilage at the edge nearest the radium tube is seen to have undergone well-marked proliferation, and below and more central to this zone is an area of degeneration (*Fig 78*). This experiment shows, moreover, the influence that the vascularity of the parts exerts upon the action of radium emanations.



FIG 78—Experiment 6. Traumatic osteoarthritis produced by radium ($\times 2$)

The Effects of the Lack of Cartilaginous Apposition and of Prolonged Cartilaginous Apposition upon Articular Cartilage—

Experiment 7—The knee of a rabbit was fully flexed and immobilized by a staple driven on either side into the lower end of the femur and upper end of the tibia—the joint not being opened. A plaster bandage was applied to complete immobilization. The rabbit was killed six weeks later and a post mortem performed. The staples had become loose and there was a considerable amount of new periosteal bone formed in the vicinity of the holes in the femur and tibia. No intra-articular adhesions between the cartilaginous surfaces were present, and the latter were everywhere normal, save over the trochlear surface of the femur which, owing to full flexion of the joint, had not been in contact with a cartilaginous surface. Here the cartilage had disappeared. Full extension was prevented by scarring of the joint capsule.

Experiment 8—The patella was completely dislocated to outer side of joint. After death ten weeks later, the articular cartilage of the patella and trochlear surface had almost entirely disappeared, evidently owing to the lack of cartilaginous apposition. Articular cartilage elsewhere was normal. No compensatory formations had as yet occurred at the margin.

These and similar experiments have an important bearing upon the pathology of osteo-arthritis, as they show that although in all probability mere immobility of a joint, provided it be healthy and that cartilaginous surfaces be in apposition, causes no degenerative changes in the latter, yet lack of cartilaginous apposition usually causes the articular cartilage to undergo transformation into connective tissue. Redfern found that the articular cartilage also underwent this change after experimental amputations through joints. It is clear that this change may occur in the human subject after amputation. *Specimens Nos 4101 and 4122*, R. C. S. Museum, show the lower ends of the femora from cases of amputation through the knee-joint. In both cases the articular cartilage is very thin, and has in places undergone transformation into connective tissue.

We frequently see examples of the same occurrence in old unreduced dislocations, both congenital and acquired, and in various deformities, and in these cases, as will be mentioned later, compensatory osteophytic formations may actually occur at the articular margins from the physiological reasons already given.

Physiological Effects of Trauma upon Articular Cartilage—Experiments were performed, of which the following is an example to ascertain whether an isolated series of traumata was in itself sufficient to produce changes in the articular cartilages.

Experiment 9—A rabbit was anesthetized and—the knee being flexed to bring the patella in contact with the trochlear surface of the femur, and the joint covered with a cloth—a series of rapid blows was applied to the patella with a hammer so that both it and the femur were subjected to repeated percussion for about two minutes. Five weeks later the rabbit was killed and the articular surfaces were found to be perfectly normal and free from fibrillation.

This experiment should be compared with *Experiment 11*, described later, where a suspension of *Streptococcus salivarius* isolated from a case of pyorrhœa was injected after percussion of a joint.

PATHOLOGY

A CHANGES IN THE ARTICULAR CARTILAGE

1 Central Part of the Articulating Area—Although not quite the earliest observable change, the most striking early departure from the normal consists in the well-known 'fibrillation' (*Figs 80 and 84*) of the central area of the articular cartilage. The poorer nutrition of the central area already referred to appears to be at least as important a factor in its earlier involvement as the greater pressure to which it is normally subjected. In the knee-joint the articular surface of the patella and trochlear surface of the femur are, in my experience, almost invariably first affected.

The term 'fibrillation' is somewhat misleading, as I am unable to agree that the change consists in the formation of true connective-tissue fibres, but find that in the cases examined by me there is a splitting of the matrix without fibrous metaplasia. Careful examination in early cases will often reveal that the first fibrils formed lie horizontally (*see extreme left of Fig 84*), and in their length occasionally far exceed the normal thickness

of articular cartilage. The staining by muci-carminic of normal cartilage reveals the reason for this disposition, since the more collagenous portions of the matrix lie superficially in horizontal strata, but are vertically disposed in the deeper portion. When vertically fractured, articular cartilage shows the same vertical strata, and my theory is that the fibrillation in osteo-arthritis is due to the persistence of the strata of more collagenous matrix. In certain cases I have observed cystic degeneration of the articular cartilage.

Case 16 (personal series) Male, age 68. The knee-joint presented well-marked signs of osteo arthritis. The portion that was examined microscopically was the posterior part of the internal condyle including the chondro-osteophyte.

In the substance of the articular cartilage is a sharply circumscribed cyst filled with finely-molecular stained material (*Fig 79*). Smaller secondary cysts lie by its side and are probably continuous. By its side is a focus showing the initial stage of cyst formation, the matrix becoming finely alveolated and the cartilage cells disappearing. In a yet earlier stage areas of the matrix lose their homogeneity and coloration, and become finely granular, the cells disappearing. In many spots liquefaction is taking place in connection with cell groups, leading to the formation of microcysts. Changes are slightly more marked in the superficial part of the cartilage, the free surface of which is here and there minutely pitted, presumably from the rupture of microcysts. The cyst lies at the base of a sessile osteophyte which is of rudimentary size and consists of cartilage superficially as far as, and including, the summit. This cartilage presents the characters of the normal articular in regard to the arrangements of the cell groups, and on the deep side presents the ordinary calcified zone, which is continuous with that of the articular cartilage of the joint. Beyond the summit the cartilage merges into a well-defined layer of connective tissue by an ordinary process of metaplasia, fibre replacing the matrix. Beneath this there is a well formed and continuous lamella of normal bone, and further outward, beyond the limit of the osteophyte, the fibrous covering is resolvable into two layers. One is of more open connective tissue furnished with delicate projecting folds covered with a well-marked layer of cells more than one in depth (synovial membrane). Beneath this the fibrous tissue is denser, lies directly on the bone and represents periosteum. The structure of the deeper part of the osteophyte is cancellous, with very open connective tissue occupying its spaces, and without any obvious fat cells.



FIG 79—Cystic degeneration of articular cartilage near base of osteophyte

Epi-articular Eechondroses—Not infrequently, the surface of the articular cartilage is rendered irregular by smooth rounded elevations due to the invasion of the deeper layers of the cartilage by vascular inroads of osteoblasts actively forming new bone, the cartilage cells simultaneously proliferating. Professor Shattock most appropriately named these nodular formations 'epi-articular eechondroses' to distinguish them from the peri-articular eechondroses at the articular edges. As the changes progress, the cartilage gradually disappears from the central articular area, revealing the subjacent bone, the changes in which will be shortly described.

2 Lateral Part of the Articulating Area—The important differences, not only in structure but in mode of nutrition, between the lateral and central portions to which we have already referred, explain the marked difference in the reaction of these parts to the cause of osteo arthritis.

The well-known lipping of the articular margins, in my experience, always succeeds the degenerative changes in the central areas. It is clear that the newly-formed cartilage is largely formed by the synovial perichondrium, since, when traced from the surface

towards the deeper parts, all the gradations from the connective-tissue cells of this perichondrium to fully-formed cartilage cells may be seen. The theory of Cornil and Ranvier which figures so largely in text-books, that the perichondrium merely prevents the proliferating cartilage cells from escaping into the joint, is not confirmed by my observations.

Some of the principal microscopic features of a chondro-osteophyte are exemplified by the following description of these formations at two different spots from the knee joint of *Case 24* (personal series).

a Section (not figured) through a portion of the posterior aspect of the internal femoral condyle, including a small recurved osteophyte. The bone is of open texture containing fatty marrow covered with cartilage in the deeper part of which there is an irregular violet zone of calcification. Near this zone there are a certain number of protrusions of cellular connective tissue, some of which also occur into the cartilage of the osteophyte itself and others towards the articulating area of the cartilage (early epi-articular formations). The osteogenetic processes of connective tissue are richly provided with cells and in one case its continuity with the bone marrow through a constricted neck was readily traceable. The marrow immediately beyond the neck on the side of the shaft is well formed adipose tissue and in the neck itself the connective tissue is being converted into fat.

The general articular cartilage shows a certain amount of horizontal fibrillation at the free surface and a little proliferation of cartilage cells.



FIG. 80.—Transverse section showing early changes in articular cartilage and structure of chondro-osteophyte. Normal vessels in synovial membrane. X = the transitional focus of connective tissue referred to in text. (Two thirds obj.)

b Another portion of articular surface including the lipped margin (*Fig. 80*) reveals the same general appearances as in the last described. However, at one spot near the base of the osteophyte there is on the deep side of the articular cartilage, a triangular focus of vascular connective tissue. The tissue is lax in character well furnished with nuclei and merges at its base into the superadjacent cartilage, from which it is clearly derived by a process of metaplasia.

Sections of synovial membrane from the immediate vicinity of the articular edge show perfectly normal structure, without any sclerosis or thrombosis of capillaries or arterioles. The villi are abnormally voluminous, the investing cells intact and devoid of any small-celled infiltration, either polymorphonuclear or lymphocytic. The vessels in the subsynovial fat are quite normal in structure, and patulous.

Although the above is the usual mode of formation of a chondro-osteophyte, occasionally a development of cartilage in a synovial fold may become superimposed upon the lateral portion of the articular cartilage with which it fuses. In rare cases the articular cartilage may be buried beneath a layer of newly formed bone formed in this manner (*Fig. 81*).

Some further changes of great interest in the bone are exemplified in *Figs 84* and *85* from *Case 27* (personal series)

A section through the patella (*Fig 84*) shows the most peripheral part of the cartilage still of normal thickness and the artificial 'flaking' due to detachment of the flat-celled layer at the surface—the cells being still living. More centrally there is a second vertical-

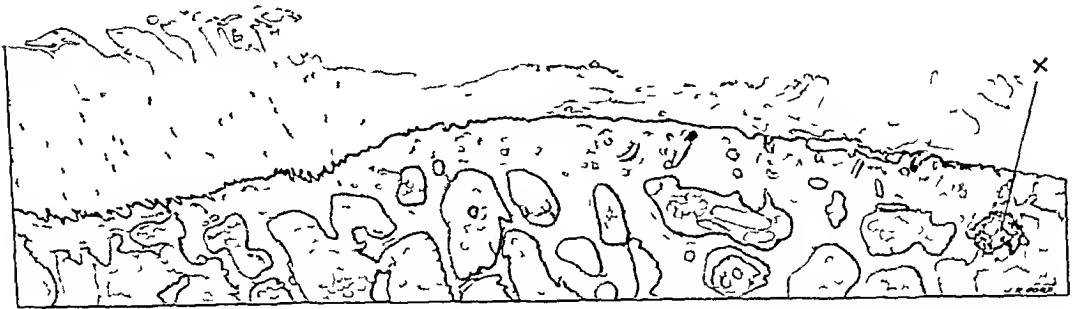


FIG 84—Transverse section of patella from *Case 27*. For description see text. X = cartilaginous nodule represented under higher magnification in *Fig 85*. (Two thirds obj.)

splitting in the cartilage, the cell nuclei retain their stain, and there is no obvious formation of fibre. Still more centralwards the cartilage becomes thinner, the cells remaining healthy, till it disappears, after breaking up into irregular fragments. The exposed articulating area here consists of very dense bone, obviously due to sclerosis of the normal cancellous tissue.

For a short way the osseous trabeculae continued from the sclerotic layer are thickened. At one spot immediately below the sclerotic zone there is a microscopic island of hyaline cartilage, and in the centre of this there is a small pseudo-cyst resulting from liquefaction—the matrix here containing no cell nuclei. Where the cartilage is wanting, or its remnants are quite detached, the sclerotic bone beneath presents a certain number of areas extending from the calcified zone, and distinguished from the proper osseous substance by their staining of a faint violet colour, and being quite homogeneous in structure, the proper bone being stained red with the eosin. In certain spots this homogeneous material is intimately mixed with the laminated structure of the bone, suggesting that it has arisen by



FIG 85—Cartilaginous nodule formed by metaplasia from bone, and itself undergoing cystic degeneration. (One-sixth obj.)

a process of degeneration occurring in the latter. The cells in the degenerate areas are unstained or stained very faintly. Here and there the more superficial parts of the degenerate tissue contain groups of cells apparently cartilaginous. Slightly below one of the extensions of degenerate tissue is the small nodule of cartilage already described.

If this small cartilaginous focus be examined under a higher power (*Fig 85*) the degenerate and more faintly staining osseous tissue may be seen above and to the right, and it is clear that the cartilage cells have arisen by a process of metaplasia from the bone corpuscles, and that the cartilaginous matrix has arisen from the bony matrix in a similar manner. The cause of the degeneration still continuing to act the cartilage itself is undergoing degeneration in the centre.

The presence of cysts in the articular cartilage has already been noted, the synovial chondromata may also undergo cystic degeneration. We thus see that there are at least three topographical varieties of cyst in osteo arthritis. There is no evidence at present that cysts of the semilunar cartilages may owe the same pathogenesis.

CHANGES IN THE SYNOVIAL MEMBRANE

In a very large proportion of cases the first naked-eye appearance of the disease occurs in the central area of the articular cartilage, and the synovial membrane very rarely shows any obvious changes until the first sign of 'lipping' appears. It will then

be noted that at these spots the membrane is thickened, and there is enlargement of the existing, with formation of new villous processes.

These changes may be quite local in the early stages. When examined during life at operation, the affected portion of the synovial membrane appears unduly vascular. Microscopical examination of the membrane in all save the very advanced cases shows that there is a general hyperplasia affecting all its elements and that the membrane and the newly-formed villi are well supplied with blood-vessels—the arterioles and capillaries being perfectly patent and showing no signs of arteriosclerosis (*Fig 86*). Specimens injected with carmine and gelatin demonstrate well that the membrane is not in a state of diminished vascularity.

In certain cases, as is well known, the enlarged villi become the seats of a formation of adipose

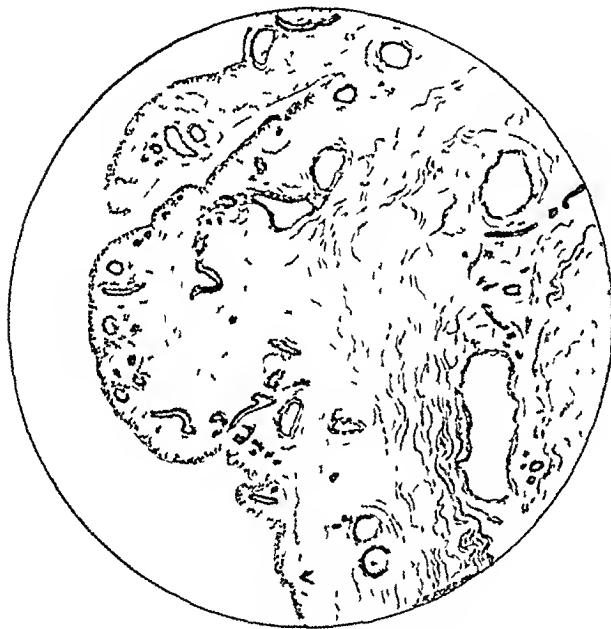


FIG 86.—Mr. Couzens' case. Vascularity of the synovial membrane in osteoarthritic knee joint such as is usually met with in the disease. The patulous vessels are represented in colour. (Two thirds obj.)

tissue (lipoma arborescens of Mallet). *Fig 87* represents a good example of this condition from St. George's Hospital Museum.

A further remarkable change is the formation of nodules of cartilage in the synovial villi. I have discussed these interesting tumours elsewhere and have named them 'synovial chondromata'. In the later stages of osteo-arthritis the hypertrophied synovial villi undergo secondary changes and the membrane becomes comparatively smooth and atrophic. In this late stage arteriosclerotic changes may sometimes be observed in the vessels of the capsule and membrane. These have been noted in one case by Hoffa and Wollenberg and more recently by Strangeways. Since, however, these vascular changes if they occur at all, occur late in the disease it is difficult to see how they can constitute an etiological factor, as these observers state.

Fig 88 shows some of the features of the later stages. The section from near the edge of the trochlear surface shows elongation of the fringes which are fibrotic, the tissue almost as far as the endothelium being dense and somewhat homogeneous. Most of the capillaries are patent in the fibrotic areas, although the fibre immediately around them is arranged somewhat conformably with the lumen. In other fringes the concentric arrangement of fibres around the capillaries obtains without any surrounding fibrosis. This striking development of fibres round the capillaries might well be termed 'pericapillaritis diffusa'. None of the fringes contains any fat. In a few spots the capillary is blocked by an associated proliferation of endothelium.

The gross changes in the capsule and intra-articular structures are too well known to require separate consideration.

Suppuration, as was originally pointed out by Sir Benjamin Brodie, is a very rare complication of osteo-arthritis. A probable explanation of this fact will be adduced below.



FIG 87—*Ipomoea arborescens* (H. J. J. & C. Corcoran, Hospital Museum)



FIG 88—Synovial membrane showing pericapillaritis diffusa (6921 St. Bart's Hospital Museum)

ETIOLOGY AND ITS RELATION TO PATHOLOGICAL DEDUCTIONS

Space will not allow a detailed consideration of the difficult problem of the etiology of osteo arthritis and necessitates a summary of main conclusions. A more complete analysis of these will be given elsewhere.

I may perhaps venture to give the following definition that I have formulated.

Osteo arthritis does not constitute a disease *sui generis* but rather the series of physiological or pathological changes that occurs in a joint when it is subjected to prolonged or oft repeated injury either mechanical or toxic, but of a moderate degree of intensity. The causes are therefore very varied and there can be little doubt that the future will bring to light additional factors in its causation of which we are at present ignorant. Osteo arthritic changes for example occur with greater frequency in certain disorders of the ductless glands such as acromegaly than in my opinion can be ascribed to mere

coincidence Whether in these cases the joint changes are due to the action of toxins formed from failure of the ductless gland to supply the necessary link in the chain of metabolic endogenous products, or whether in some way the resistance of the joint to bacterial toxins is lowered, it is impossible at present to state

The relation of osteo-arthritis to the group of auto-intoxications due to defects in the excretory apparatus or the accumulation in the body of products of normal metabolism is still undecided In spite of these undecided factors, there emerge two groups concerning the etiology of which we have a little more evidence The following preliminary classification is therefore adopted
(A) Traumatic osteo-arthritis, (B) Osteo-arthritis due to bacterial toxins which are (a) formed locally, (b) brought from some distant focus

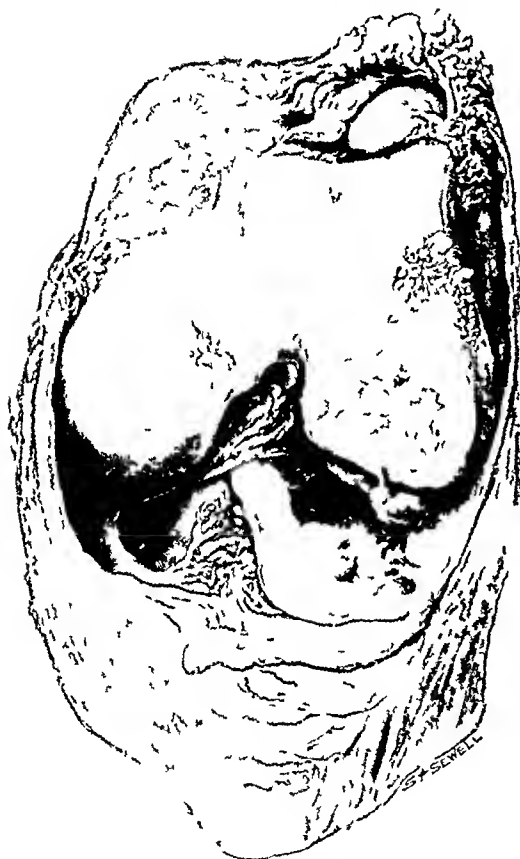


FIG. 89.—Osteo-arthritis and fracture dislocation of external semilunar cartilage, a loose body of the classical type is also present in upper part of joint (7091, Middlesex Hospital Museum)

Group A Traumatic or Localized Osteo-arthritis—Examples of this large group occur very frequently, and have moreover, considerable medico-legal importance In many cases it is difficult to be certain whether we are dealing with a case of true traumatic osteo-arthritis or whether the injury has lowered the resistance of the joint structures and caused the latter to become the site of action of bacterial toxins It must not be forgotten that although articular cartilage is devoid of vessels, yet it exhibits the cellular response which constitutes one of the principal phenomena of inflammation, and that the latter may be brought about by mechanical injury quite apart from the action of toxins True traumatic osteo-arthritis is usually distinguished by the fact that the pathological changes are in most cases localized for a considerable period to that part of the joint which is subjected to the greatest degree of mechanical trauma Moreover an important etiological factor lies in the fact that the traumata are oft-repeated and spread over a long period of time Clinical and experimental observations cause me to doubt whether an isolated contusion of a joint is in itself sufficient to cause true

traumatic osteo-arthritis, although it may undoubtedly be a predisposing factor

Classification of traumatic osteo-arthritis—Among the causes may be enumerated—

- 1 The presence within the joint of a loose body or other localized source of irritation
- 2 Fractures involving joint surfaces or of the adjacent bone that bring about altered alignment of these
- 3 Altered articular alignment from disease of the limb bones with consequent deformity
- 4 Localized increase of articular stress of an occupational origin (Arbuthnot Lane)
- 5 Osteo-arthritis in false joints
- 6 Abnormal joint mobility, from rupture or stretching of capsular or intra-articular ligaments
- 7 The repeated intra-articular hæmorrhages of hæmophilia

Examples of these varieties are of frequent occurrence, and space does not permit then detailed discussion. It can hardly be sufficiently emphasized, however that in many cases the occurrence of the traumatic form is evidence of improper or neglected treatment.

Fig 89 represents a knee-joint from the Bland-Sutton Institute of Pathology of the Middlesex Hospital. A loose body of the traumatic type derived from the articular surface of the patella which was fractured some years previously, lies immediately above the outer part of the trochlear surface of the femur. Secondly, there is a marked displacement forwards of the posterior end of the external semilunar cartilage which has become twisted and adherent to the anterior end. The osteo-arthritic changes are largely confined to the outer part of the joint.

It is not uncommon to find changes in the articular cartilage, consisting in fibrillation, erosion, or nodular eminences beneath an abnormally mobile or damaged semilunar cartilage of long standing.

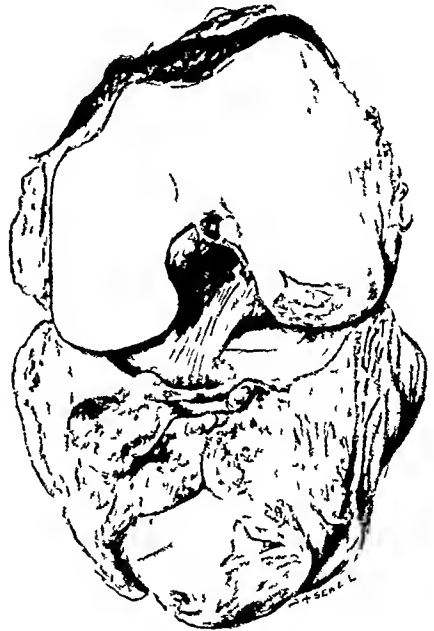


FIG 90—Early osteo arthritic changes in knee-joint from case of hemophilia (740C St Bart's Hospital Museum)



FIG 91—Osteo-arthritis occurring after several attacks of acute rheumatism (4111 Guy's Hospital Museum)

These changes are to be distinguished carefully from those arising after operations on the semilunar cartilages in which, either from division of the internal lateral ligament or faulty after-treatment, a condition of abnormal joint mobility has arisen. Clinical and experimental experience convinces me that when loose bodies in joints are smooth, encapsulated, or situated in some part of the joint where they are unable to damage the articular cartilage, osteo-arthritic changes are unlikely to occur.

The following experiment demonstrates the latter points—

Experiment 10—The right knee joint of a rabbit was opened by a vertical incision on the inner side, and a small pellet of sterile wool was inserted into the supra patellar pouch. In addition, three small sterile leaden shot were introduced. The wound was sutured in two layers and a collodion dressing applied.

The after history was quite uneventful, the animal used the limb normally and there was no limp. Seventeen weeks later, on opening the joint, it was seen that the pellet of wool which had remained in the supra patellar pouch was surrounded by a smooth and glistening sheath of connective

tissue. The leaden shot were lying perfectly free and unaltered in the lower and front part of the joint. Careful examination failed to reveal any fibrillation or other abnormal changes in the articular cartilage. This specimen is now in the Museum of the Royal College of Surgeons.

When a traumatic loose body (i.e. a detached portion of the articular surface) is quite free, it occasionally gives rise to generalized osteo-arthritic changes. These are preceded by oft-repeated attacks of synovitis, and the articular cartilage participates in the inflammatory reaction caused by the loose body becoming caught between the articular surfaces. In some cases these osteoarthritic changes are associated with the formation of synovial chondromata, and I have observed the association in the same joint of a traumatic loose body and of detached synovial chondromata.

Fig 90 (Specimen 710c St. Barts Hosp. Museum) shows early osteoarthritic changes in the left knee-joint from a fatal case of hæmophilia—a boy, age 13. At the under surface of the external condyle of the femur fibrillation and wearing away of the cartilage have occurred. Similar changes are also present in the articular surface of the patella and the synovial membrane is markedly stained. In the right knee (not figured) the changes are far more advanced, and osteophytes are present. There can be little doubt that these changes in hæmophilic joints are directly due to the mechanical irritation of the articular cartilage by blood.

Group B Osteo-arthritis due to Bacterial Toxins —

a Osteo arthritis occurring in the more chronic forms of the so called specific infections, such as typhoid fever, pneumonia, dysentery, gonorrhoea, syphilis, etc.

My personal observations confirm those made by most other workers, that in these chronic joint affections it is rarely possible to isolate organisms from the joint fluid. However, it is illogical to argue from this that the joint affections are unconnected with the organism which has given rise to an existing or recent infection.

Two alternative explanations of this absence of organisms from the joint fluid may be given. (1) The specialized synovial cells which are disposed in many layers, particularly near the articular margins, form a barrier effectually shutting off organisms from the joint cavity, save in the more acute cases. The toxins elaborated by these organisms gain access, however, to the synovial fluid. (2) The bacterial toxins are brought to the joint from some distant focus. This appears the more likely hypothesis in most cases, the reason for this view will be adduced later.

As an example of the subdivision under consideration may be cited the occurrence of osteo arthritis after acute rheumatism.

Fig 91 (Specimen 4513 Guy's Hosp. Path. Museum) depicts a knee-joint from a case of rheumatism, and shows both acute and chronic disease. The articular cartilage is fibrillated and softer than normal, and in addition there is some ulceration of the articular cartilage of the femur and patella. The margins of the condyles are slightly bipped. The synovial membrane is thickened and covered with polypoid outgrowths. The ulceration of the cartilage is probably of recent date, and the fibrillation, synovial overgrowth, and bipping are osteo-arthritic changes caused by repeated attacks of acute rheumatism. It is from a woman, age 25, who was admitted for acute rheumatism and died in the hospital. At the autopsy, both knees were found to contain opalescent fluid in which were masses of fibrin. Myocarditis and valvular disease of the heart were present.

In this connection it is of interest to note that Poynton and Paine and Beattie have produced osteoarthritic lesions experimentally with organisms isolated from cases of rheumatism.

b The so-called 'idiopathic' 'spontaneous', or 'senile' osteo arthritis

This type is extremely common in persons who have passed the meridian of life, and cases abound in every out-patient department. There is a school which ascribes such forms of osteo-arthritis occurring in elderly persons to the so called 'senile degeneration' (*vide* the term 'degenerative arthritis'). Hoffa and Wollenberg have suggested that the changes might be due to deficient nutrition of the joint structures brought about by endarteritis obliterans of the nutrient vessels. More recently, Strangeways of Cambridge has advanced the theory that the changes are due to alteration in the nutritive value of the synovial fluid, which is brought about by arteriosclerosis of the vessels of the joint capsule.

DEDUCTIONS TO BE DRAWN AS TO THE ETIOLOGY OF THE 'IDIOPATHIC' OR 'SENILE' FORM OF OSTEO-ARTHRITIS FROM THE PATHOLOGICAL AND CLINICAL DATA, WITH AN ACCOUNT OF SOME EXPERIMENTAL OBSERVATIONS

a Deductions from the Morbid Histology of the Affected Joints and from the Composition of the Synovial Fluid—

1 My analyses of synovial fluid from osteo-arthritis joints reveal the fact that it is actually richer in protein content than normal synovial fluid. Secondly, on the theory of altered nutrition by the synovial fluid, the changes should take place in those parts of the cartilage most remote from its access, whereas the reverse is the case.

2 Although the ground substance of the articular cartilage, particularly of the central area, certainly degenerates, yet the cartilage cells usually show no sign of degeneration, and may actually proliferate. This proliferation is particularly well-marked in the lateral parts of the articular cartilage where chondro-osteophytes, often of large size, may be formed. This proliferation is *difficult to reconcile with the theories of senile degeneration or of diminished nutrition*.

3 The changes in the bone are at first hyperplastic, and it is only at a later period that the newly-formed bone becomes atrophic, and its cancellous spaces of open and fatty texture.

4 The changes in the synovial membrane are at first *hyperplastic rather than degenerative*. The marked increase of synovial villi, with formation in some cases of synovial chondromata, is extremely difficult to reconcile with any form of senile degeneration.

5 The synovial membrane in all the earlier cases examined was found to be highly vascular, the larger vessels and capillaries being patent and presenting no sign of endarteritis obliterans. There is evidence, however, that in the later stages some endarteritis may be present, and the synovial membrane admittedly becomes atrophic.

6 The joint changes bear no constant relation to age. Similar pathological appearances may occur in middle-aged or young individuals, in whom these 'senile' changes may be very marked. Furthermore, in very aged individuals the lesions of osteo-arthritis may be absent.

7 The conclusion to be drawn from the morbid histology strongly favours the view that the joint structures *are acted upon by toxic substances*, which, in the poorly-nourished central area of the articular cartilage, bring about degeneration of the ground substance, and elsewhere proliferation. The latter process is followed later by degeneration from the continued action of the toxin. The anatomical position of the lesions strongly suggests that the circulus articularis vasculosus is the principal vascular route through which the toxins reach the joint.

8 The usual absence of small-cell infiltration in the various joint structures and of micro-organisms in the synovial fluid suggests that the toxic substances are not formed by bacteria *in situ* but are brought from some other part of the body.

*b Deductions upon the Possible Origin of the Toxic Substances from the General Pathological Appearances as Revealed Post Mortem—*It was thought that a number of investigations of the general pathological appearances might throw some light upon the source of the toxic substances. In this part of the research it is my pleasant duty to acknowledge the help and facilities accorded me by Sir Frederick Andrewes and Dr Spilsbury.

From the subjoined tables it will be seen that—

Out of seventeen cases in *Table I* (slight osteo arthritis) potential foci of infection were present in fifteen.

Out of six cases in *Table II* (moderate degree of osteo arthritis), in four there were distinct foci of potential infection and in two the evidence points to intestinal toxæmia. In all six cases well marked co-existent lesions of an infective or toxic nature were present.

Out of three cases in *Table III* (advanced osteo arthritis), in two potential

infective foci were present and in all three various co-existent lesions—probably of infective or toxic origin

We thus see that the evidence derived from the general post-mortem pathological appearances in cases of osteo-arthritis certainly lends support to the theory that the disease may be caused by the action, amongst others, of toxic substances elaborated in chronic infective foci. It should be added that since it is not usual to pay particular attention to the teeth, gums, tonsils, or accessory nasal sinuses in ordinary post-mortem examinations, these possible sources of toxic absorption must be ruled out of count in the subjoined table. Moreover, intestinal toxæmia may be present without any very striking naked-eye pathological changes. It is therefore possible that many of the potential infective foci found were themselves secondary to intestinal infection.

Table I—SICKLE CELL ANEMIA

NO.	SEX	AGE	CAUSE OF DEATH	POTENTIAL INFECTIVE FOCUS OR FOCI	COEXISTENT LESIONS PROBABLE OF INFECTIVE OR TOXIC NATURE
1	M	67	Cerebral embolism	Chronic empyema evidence of old ulceration of stomach	Atheroma of coronary arteries fibrosis of pancreas
2	M	50	Fracture of base of skull	Tuberculous foci both lungs	Cirrhosis of liver and kidneys
3	M	75	Strangulated inguinal hernia	? Intestinal toxæmia	Granular kidneys patches of atheroma of aorta
4	M	56	General peritonitis	Chronic cholecystitis with biliary calculus	Cirrhotic liver granular kidneys
5	M	56	Lobar pneumonia	? Intestinal toxæmia	Chronic pancreatitis
6	M	58	Arteriosclerosis	Gastric ulcer	Cirrhotic liver and granular kidneys atheroma of aorta
7	M	57	Aneurysm of aortic arch	Syphilitic infection	Syphilitic aortitis arteriosclerosis
8	M	67	Permeious anemia	Superficial ulceration of large bowel ? Intestinal toxæmia	Usual signs plus thickening of coronary arteries
9	M	48	Carcinoma of stomach	Toxic absorption from growth	No general P.M. performed
10	M	47	Perforated gastric ulcer	Large chronic ulcer of stomach	No general P.M. performed
11	M	28	Infective endocarditis		Chronic parenchymatous nephritis mitral and aortic endocarditis infarcts spleen and kidney etc.
12	M	66	Carcinoma of the œsophagus	Toxic absorption from growth	No general P.M. performed
13	F	34	Carcinoma of stomach	Toxic absorption from growth	Kidneys congested with cloudy swelling
14	M	40	Pulmonary necrosis	Toxic absorption from growth	No general P.M. performed
15	F	39	Lobar pneumonia	Chronic bronchiectasis right lung	No general P.M. performed
16	M	39	Carcinoma of stomach	Toxic absorption from growth	No general P.M. performed
17	F	16	Septicæmia	Suppurative arthritis left shoulder	Lungs congested and œdematous

Table II—MODERATE DEGREE OF OSTEO-ARTHRITIS

NO	SEX	AGE	CAUSE OF DEATH	POTENTIAL INFECTIVE FOCUS	COMPLICATING LESIONS, PROBABLY OF INFECTIVE OR TOXIC NATURE
1	M	59	Phthisis	Both lungs full of abscesses	Atheroma of coronary arteries and aorta
2	M	57	Cirrhosis of liver	Intestinal tract	Cirrhosis of liver and pancreas
3	F	49	Uremia	Chronic suppuration in accessory sinuses	Kidneys sclerotic Old perisplenitis Tricuspid and mitral valves thickened
4	F	66	Cholelithiasis	Biliary tract	Pancreatitis Abundant growth of <i>B. coli</i> from urine
5	F	48	Septicemia	Wide spread chronic dermatitis	Cloudy swelling of kidneys Fatty degeneration heart and liver
6	M	51	Carcinoma of head of pancreas	Intestinal tract	Remainder of pancreas hard and fibrotic—obstructive biliary cirrhosis

Table III—ADVANCED OSTEO-ARTHRITIS

NO	SEX	AGE	CAUSE OF DEATH	POTENTIAL INFECTIVE FOCUS	COMPLICATING LESIONS, PROBABLY OF INFECTIVE OR TOXIC NATURE
1	M	68	Cerebral hemorrhage	—	Atheroma of cerebral renal, and coronary arteries, and of aorta
2	M	65	Fractured base	Chronic cholecystitis	Vegetations on aortic valves Atheroma of aorta Evidence of old peritonitis
3	M	49	Uremia	Chronic cystitis Suppuration in prostate	Interstitial nephritis Atheroma of aorta

The frequency with which atheroma and arteriosclerosis were present in the above series is of great interest, and suggests that they have the same cause as the osteo-arthritis changes. Furthermore, as a general rule it was found that the degree of osteo-arthritis present was proportionate to the extent of the arteriosclerotic changes. The theory that arteriosclerosis is primarily inflammatory rather than degenerative we owe to Virchow and the evidence in favour of his view is strong, although, as in osteo-arthritis, there is an absence of small-cell infiltration. It is probable that old age is a factor, as in osteo-arthritis, only through virtue of the fact that it allows time for chronic bacterial infection gradually to break down local powers of resistance.

c Deductions from Clinical Data—Space will not permit the enumeration of case-histories but compels me to summarize my conclusions from a critical study and analysis of a large number of hospital and private patients. In at least 95 per cent of cases of osteo-arthritis I have been able to satisfy myself that a definite focus or foci of toxic absorption were present.

I am unable to find records of a single case where treatment directed towards the focus or foci in question has not produced amelioration of the joint condition and in many cases cure has resulted. A careful analysis of my personal cases and of cases published by other investigators—including those of Sir Arbuthnot Lane* and Sir Kenneth Gairdner

* I am indebted to Sir Arbuthnot Lane and to Dr. Mutch for kind help in this aspect of the research.

—convince me that the arthritis on the clinical evidence must be considered as the result of the infective condition and that this co-existence and the effects of treatment cannot be dismissed by any rational person as a mere fortuitous coincidence.

The possible foci of toxic absorption are very numerous, and my experiments upon animals led me to believe that the joint resistance is gradually broken down. It is this prolonged resistance of the joint which probably accounts for the fact that osteo-arthritis is more common in those past the meridian of life.

d Deductions from Experimental Observations—It is obvious that if we are able to produce osteo-arthritis experimentally by bacterial toxins, and particularly if the bacteria are obtained from an obvious focus of infection in a patient suffering from osteo-arthritis, this evidence must lend strong support to the theory of causation of the latter by bacterial toxins.

The following typical experiment of mine may be quoted—

Experiment 11—The *Streptococcus salivarius* was isolated by Dr Standish from the teeth sockets of a male patient, age 65, with marked pyorrheal alveolitis, who also had osteo-arthritis of the left hip. A broth culture of the organism was prepared. Of this culture 1 c.c. was injected into the right knee, and half this quantity into the left knee, of a rabbit. The left knee had been previously subjected to percussion for two minutes with a mallet. Six weeks later the animal was killed. The left knee (which it is to be noted had received only half the amount of organisms received by the right, but which had been subjected to percussion) was filled with thick creamy pus, the synovial membrane was converted into granulation tissue, and some absorption of the articular cartilage had occurred. No true osteophytes were present. The right knee joint presented unequivocal signs of osteo-arthritis.

This experiment demonstrates also the important part which trauma may play in lowering the resistance of the joint to infection and affords some explanation of the frequency with which the onset of osteo-arthritis is preceded by trauma.

As is well known, the joint fluid in osteo-arthritis is in a very large proportion of cases, sterile. I have, however, found that this sterile fluid when injected into the joints of rabbits induces degenerative changes in the articular cartilage, thus demonstrating that the fluid from osteo-arthritic joints contains toxic substances.

Experiment 12—From the knee joint of a case of early osteo-arthritis with effusion, 2 c.c. of sterile synovial fluid were injected into the right knee joint of a rabbit, and 15 c.c. into the left. Six days later it was noted that the temperature was raised over both joints, and some limitation of flexion was present from muscular spasm. Thus, however, soon disappeared, and the joints appeared normal. Four months later, however, on examination the joints showed well marked superficial erosion of the articular cartilage at the junction of the trochlear and condylar portions of the articular surface.

The following experiment was performed to ascertain the reaction of the joint to metabolic poisons formed in the intestinal canal upon the organic constituents of the intestinal contents. In the bowel tyrosin is first changed to highly poisonous hydroxy-phenylethylamine (tyramine acid phosphate) and ultimately to the relatively innocuous phenol.

Similarly β -iminazolyethylamine (histamine phosphate) is formed from histidin.

Experiment 13—Into the right knee-joint of a rabbit 0.5 gram of histamine phosphate dissolved in 1 c.c. of sterile water was injected and 0.227 gram of tyramine acid phosphate in a similar quantity of sterile water was injected into the left knee joint. It should be noted that 0.001 gram hypodermically is the human dose of the former and 0.02 gram of the latter.

No reaction of any kind occurred in the joints, nor was any constitutional reaction observed.

Osteo-arthritis Occurring in Diseases of the Central System, such as Tabes and Syringomyelia—As is well known, in these diseases the joints may be the seat of pathological changes, which save that all the processes tend eventually to become exaggerated, are, as Sir Frederick Eve pointed out, similar in all respects to those occurring in osteo-arthritis. It seems highly probable that the osteo-arthritic changes are not due directly

to the disease of the central nervous system, but that the latter in some way leads to the increased rapidity and extent of these changes

Osteo-arthritis Occurring in Chronic Gout—Microscopical examination of the articular cartilage in gout does not lend support to the view that the osteo-arthritic changes are due to the irritation induced by the presence of crystals of sodium bicarbonate, for there is very little proliferation of the cartilage cells in their vicinity—we are not thus faced with a form of traumatic osteo arthritis. The actual etiology of gout is still unsettled, but on the analogy of the types of osteo-arthritis already passed under consideration, it would appear that the joint changes occurring in the chronic form of the disease are brought about by the action of bacterial toxins

SYMPTOMATOLOGY AND DIAGNOSIS

Although, as we have already noted, osteo-arthritis particularly when of the traumatic and infective types, may occur in young persons, yet the subjects it selects are usually middle-aged or elderly. In my experience males are slightly more liable to the condition than females. The traumatic form usually, but not invariably, affects a single joint, whereas the infective or toxic form may be uni- or polyarticular. The latter variety commonly commences in the hands and feet, where the terminal interphalangeal joints are usually first affected, often with formation of Heberden's nodes. The onset is usually slow insidious, progressive, and unassociated with raised temperature or marked constitutional symptoms. Aching in one or more joints after use, often associated with a slight degree of swelling, is generally the first symptom. The pain and swelling gradually increase, and if examined at the early stage it is often possible to find slight increase of temperature over the joint and a little painful limitation of movement by muscular spasm. The presence of fluid may not infrequently be detected, and in certain cases fluid may be present in marked quantity, not only in the joint itself, but in bursæ communicating therewith, and occasionally in bursæ and tendon sheaths quite unconnected with the joint cavity. In a series of investigations of the synovial fluid from these cases in which Dr Arthur Davies, pathologist to the Dreadnought Hospital, Greenwich, has rendered invaluable assistance, we have not succeeded in discovering the presence of bacteria, but from the observations already made, too much weight must not be placed upon this negative evidence. The fluid, as has been stated, is rich in albumin, and differs thus markedly from normal synovial fluid.

In some cases a somewhat rapid effusion of fluid may form the first sign of the disease. This early heat, pain, and fluid effusion appear to point strongly to the inflammatory, rather than to the degenerative, origin of osteo arthritis.

The aching and pain in the joint after use are rarely sufficient to prevent the patient from following his occupation in the early stages. Stiffness after rest, and particularly first thing in the morning, become marked features, although this symptom in itself rarely causes the patient to seek relief. Thickening of the synovial membrane or capsule is not a marked feature at this stage. After a variable period, lifting of the articular margin may be distinctly felt, the earliest lifting, as it consists of cartilage only, may not be apparent on x-ray examination. The synovial membrane can now be felt to be thickened particularly round the patella and trochlear surface of the femur, and loose bodies such as chondromata growing therefrom may be detected. There is 'snowball crunching' crepitus on movement from the mutual apposition of thickened synovial fringes.

In the later stages when the articular cartilage over the central area has been worn away and the bone has become exposed pain becomes a more marked feature. The patient often complains of a constant gnawing pain as if the 'bones are grinding together'—indeed they are. The constant pain worse on exercise, is often of such an exhausting nature that a patient may rapidly lose weight become markedly neurasthenic and his existence be sadly embittered. On the other hand symptoms may be remarkably slight. At this stage osteophytic outgrowths may be very marked and there is usually

harsh grating on movement (although when ebunation occurs movement becomes smoother), associated with shortening lateral mobility, and various kinds of deformity. At this stage there is usually an absence of fluid in the joint ('arthritis secche').

The rarefaction, which, as we have already described, occurs in the cancellous tissue of the articular extremities shows up in a conspicuous manner on x-ray examination which may reveal pseudo-cystic spaces traversed by attenuated bony trabeculae.

Differential Diagnosis from Rheumatoid Arthritis—In rheumatoid arthritis we have the clinical and pathological picture of a more marked inflammatory process than is the case in osteo arthritis. This inflammatory process brings about a reflex muscular spasm which tends in the more acute types to cause a considerable degree of limitation of joint movement. Owing to the immobility of the joint, no obstacle is placed in the way of the outgrowth of a synovial pannus which gradually extends over the surface of the articular cartilage. There can be little doubt that the vitality of the cartilage is seriously interfered with through the action of toxins, and that the advancing synovial pannus of granulation tissue serves the purpose of replacing the degenerate cartilage. It is easy to see that the end-result of this process is usually intra-articular ankylosis.

In the more chronic process of osteo arthritis muscular spasm is slight or absent, and the continued movements prevent the pannus of granulation tissue from the synovial membrane from encroaching upon the surface of the articular cartilage, just as the movements of the foetus before birth gradually disperse the connective tissue covering the articular cartilage, true intra-articular ankylosis therefore occurs rarely, if ever.

In rheumatoid arthritis, the swelling of the synovial membrane and capsule is more marked, and causes the typical spindle-shaped swelling of the joint. The disease occurs more commonly in women between the ages of 20 and 40, and usually several joints are affected. Every stage of acuteness may be seen, and constitutional symptoms are often present, such as anæmia and wasting, also vasomotor changes such as sweating, coldness and glossiness of the hands or feet, or tingling, numbness and a sensation of pins and needles in the same situations. Associated enlargement of lymphatic glands and spleen may occur in children (Still's disease), and not infrequently this glandular enlargement may be detected in adults. The prolonged muscular spasm causes atrophy of the tissues surrounding the joints, including the skin, hence the name 'atrophia' which is sometimes given to the disease or group of diseases.

TREATMENT *

In the traumatic group, prevention is naturally better than cure, and in many cases the occurrence of this variety is due to faulty treatment. However, as this form is usually localized, appropriate treatment of the deformity or other source of irritation where practicable may stop the progress of the disease.

I would adduce the following general principles of treatment, which are particularly applicable to the earlier and active stages of the disease, and for the infective or toxic group, including the 'senile' variety—

(1) *Eradication, as far as possible, of any focus or foci of toxic absorption, including measures that prevent the formation or assist the elimination of systemic toxins.* (2) *The cessation, particularly in un articular cases and in the lower extremity, of pressure between diseased articular surfaces.* (3) *Local treatment to the joints themselves to stimulate defensive reaction of articular elements, and to prevent ankylosis.* (4) *Dietetic and medicinal treatment.*

It is desired to emphasize strongly that it is in the early stages, before serious structural changes have occurred in the affected joints, that treatment is often of great benefit and cure may result. The pessimistic or *laissez-faire* attitude prevalent concerning the treatment of osteo arthritis appears to be unjustified, and exists because, from ignorance of the cause, the joints have been allowed to drift to advanced structural change.

* I am indebted to Sir Robert Jones for kind suggestions in the preparation of this section.

1 The Eradication of Foci of Toxic Absorption—The possible foci of toxic absorption are very numerous, and several may co-exist. A careful and exhaustive examination of the whole patient is necessary in every case, and should particularly include the accessory sinuses and the whole of the alimentary, respiratory, and genito-urinary systems.

Co-operation in this search between surgeon, physician, and bacteriologist is absolutely essential, and it is this lack of co-operation that is responsible for so many failures.

In my series of cases of osteo-arthritis, failure to discover such foci of toxic absorption or evidence of their previous existence, was rare. In many cases the adherence to this principle combined with vigorous local treatment has led to cure. Although I attach importance to the presence of pyorrhœa alveolaris, yet it appears probable that in most cases the condition is associated with bacterial infection of other parts of the intestinal tract by organisms, particularly streptococci, which have escaped destruction by the gastric juice.

In some of these cases, as Sir Arbuthnot Lane and his co-workers have shown, intestinal stasis may be demonstrated, and in others the feces contain pathogenic bacteria. It is my practice in all cases where marked pyorrhœa is present to investigate as fully as possible the condition of the whole alimentary tract. Toxic absorption from the latter constitutes the commonest mode of origin of osteo-arthritis. In women the uterus and its adnexa should always be investigated and inquiry made as to menstrual irregularities or the presence of discharge.

Simultaneously with treatment directed towards the eradication of foci of toxic absorption, the principle of treatment under discussion should include measures that assist the elimination of toxic substances by the skin, bowels, and kidneys, and the raising of the natural powers of resistance of the patient by every means within our reach. The latter may profitably include the use of autogenous vaccines.

2 The Diminution of Intra-articular Pressure—*Rationale*—The articular cartilage, one of the most important functions of which is to preserve the subarticular bone from friction, is being acted upon by toxic substances, and if subjected to pressure and friction, must become worn away. Furthermore, the cancellous bone is atrophic and unable to stand normal pressures. Every effort should therefore be made to diminish articular pressure, particularly in the lower extremity, in the early stages, and if the toxic focus is dealt with vigorously at this stage, a cure may sometimes be anticipated.

In cases with somewhat acute onset it is justifiable to immobilize the limb in plaster, it first to attain this end, the joint being placed in the position which experience shows to be best should ankylosis occur.

In cases of average severity, the principle may be attained in the lower extremity by the wearing of a splint which prevents or minimizes intra-articular friction, but allows the patient to take a moderate amount of exercise, for this improves the general condition of the patient and tends to prevent muscular wasting. The exercise should be carefully supervised as if carried to excess it may be harmful.

In the lower extremity the principle of extension or of diminution of intra-articular pressure by keeping the joint surfaces apart, combined with movement, although ideal theoretically usually necessitates expensive apparatus, and it is doubtful whether the joint surfaces can be separated sufficiently without exercising strain upon the capsular ligaments and synovial membrane which are themselves often diseased. However, apart altogether from the question of combining extension with movement there is no doubt that the wearing of a comfortable light, and well-fitting support such as a Thomas ciliper splint or moulded leather support which keeps the joint in the position of greatest use to the patient must markedly diminish intra-articular friction and usually brings about considerable relief. Pressure and friction are naturally less potent in the upper extremity, but the same principles may be applied.

By this combination of diminution of intra-articular friction and treatment of foci of toxic absorption very marked improvement may be expected in early stages. Unfortunately owing to the moderate severity of all the symptoms the patient does not often

seek relief until grave structural changes have taken place in the joint. The problem of treatment is then a more difficult one and the results are by no means so satisfactory.

The principle of diminution of intra-articular friction and of weight-bearing in the lower extremity remains the same.

Broadly speaking we may say that the principle may be achieved either by operative or non-operative measures and that we may aim either at ankylosis or at the retention of a movable joint. The particular measures to be adopted depend on many different factors, and each case must be carefully considered on its own merits. A fairly firm extra-articular fibrous ankylosis may usually be attained by fixation in the optimum position by a splint. If we desire to retain movement, short of drastic measures, the method of combining extension with movement mentioned above may be adopted. Before a splint can be applied to bring this about manipulations to stretch adhesions, tenotomies, and in some cases the chiselling away of obstructing osteophytes may be necessary. Even if it is decided to aim at ankylosis these preliminaries may be necessary in order to obtain the best position. A large number of these cases are painful because of the presence of adhesions in the joint. If an anæsthetic is given and the adhesions are broken down, and if movements are practised aided by a masseur an improved range of movement with alleviation of pain often ensues and this improvement is maintained for a considerable time. These manipulations should always be performed by a qualified medical man, and never under any circumstances by the instrument maker. Experience shows this caution to be very necessary. When the splint is applied the patient no longer hesitates to walk through fear of pain and his general condition may markedly improve.

There is reason to believe that if the toxic focus is properly dealt with the process of absorption may cease, the exposed bone although it never becomes covered anew with articular cartilage, yet becomes sclerosed hard and polished and a natural cure may be said to have occurred.

Operative measures are indicated in acute cases with severe pain or in young or middle-aged persons in whom the disease appears to be no longer active but has left a painful and deformed joint or one in which movement is limited. It is far too frequently overlooked that if a focus of toxic absorption exists any benefit derived from operation must be of a temporary nature only unless this is accompanied or preceded by a successful attack upon the focus.

The operative measures vary considerably and every case should be carefully judged on its own merits taking into consideration the patient's age, occupation and general constitution. The results are sometimes disappointing, and this should be explained to the patient whose personal desires, in addition, should be carefully considered. For example the pros and cons of ankylosis or of an attempt to obtain a movable joint should be frankly discussed.

Arthroplasty or the interposition of pedicled flaps of fascia or other substances between joint surfaces denuded of articular cartilage is sometimes performed in the case of the hip, elbow, patellofemoral joint, etc. In certain cases marked improvement occurs especially if any toxic focus is sought out and successfully treated or the process has come to an end. Otherwise it would appear that the fascial flap must undergo the same fate as we have seen occurs to other intra-articular connective tissue structures.

In a recent discussion on the treatment of osteo arthritis of the hip-joint at a meeting of the British Orthopædic Association it was almost unanimously agreed that the operation of arthroplasty of this joint was disappointing in its after-results. In the light of the pathological data given above the reason for this seems clear. Arthroplasty of the hip in no way diminishes weight-bearing and it is the weight of the body transmitted through the trochanteric articular ends of the bones that causes a continuance of the symptoms. The operation devised by Sir Robert Jones often proves very beneficial in old people who are unable to stand the shock associated with eversion. It is quickly performed and gives rise to very little shock.

Removal of Osteophytes—The ruthless chiselling away of osteophytes is to be deprecated since we have seen that they are compensatory developments which often play a

useful role. The rare indications for removal of osteophytes are (1) When they interfere with movement, (2) When they cause severe pain by pressure on an adjacent nerve (3) When they are themselves subjected to painful pressure.

Handley's operation of cheilotomy is especially indicated in comparatively young persons whose symptoms are not acute, but in whom movement is markedly restricted by osteophytic formations and who should be given the chance of a more movable joint short of the more drastic measures of arthroplasty or excision. An important practical point to bear in mind in these cases is that there is usually adaptive shortening of the capsule and extra-articular structures, and removal of the osteophytes does not in itself in my experience usually restore full movement although pain is markedly lessened.

Excision is particularly indicated in suitable cases where pain is very acute. It may be performed either to obtain fixation or movement. In the former case it must be remembered that although the cancellous tissue of the bony extremities is of very open and fatty texture, yet good union usually occurs.

Excision of the hip is rarely indicated in elderly persons, since the turning out of the head of the femur is associated with a considerable degree of shock.

In young or middle-aged persons the operation is indicated where pain is a marked symptom. In the past the operation has been perhaps somewhat unjustly condemned. If however, proper after-treatment is adopted, and the toxic focus suitably treated, there appears to be no reason to expect in the new joint the instability and recurrence of the disease which has been often noted in the past.

Lithodesis.—The results of this procedure particularly in the case of the hip have been somewhat disappointing owing to frequent failure to obtain union. I believe that the disappointing results can be largely explained on pathological grounds since, as we have seen, the subarticular layer of bone is sclerosed and does not therefore readily unite with the corresponding opposed layer. The turning out of the femoral head is associated with a considerable degree of shock, which in itself is a drawback of the operation, particularly in elderly people.

Local Treatment of the Joints Themselves.—It can hardly be sufficiently emphasized that unless the first two principles are adhered to no great or lasting benefit may be derived from local measures. The latter form a useful adjunct but used by themselves almost invariably lead to disappointment. Unfortunately, it is only too common to see cases that have tried every form of local therapy and have travelled from one hydrotherapeutic establishment to another, and during the whole period no attention has been paid to the first two all-important principles.

Radiant heat, electricity, local baths, ionization, massage and movements both active and passive are often of great value since the stagnation of circulation that favours toxic action and inhibits the action of antibodies is prevented. Furthermore in all cases of osteoarthritis unless we are definitely aiming at ankylosis the joint or joints should be put through their full range of movement each day, for by this precaution ankylosis, deformity and muscular wasting may be largely prevented and in addition interference with movement by osteophytes is thus avoided.

The fault of some in local measures cannot be better illustrated than by the fact that in a modern work on osteoarthritis the first two principles that I have mentioned are ignored altogether and the book ends with an appeal in support of the treatment at a well known hydrotherapeutic establishment. The treatment recommended for bony nodes of the Heberden type is that they should be wrapped in lint soaked in a saturated solution of sodium chloride. The author naïvely remarks that the method must be used for a considerable time if good results are to be obtained.

Dietetic and Medicinal Treatment.—Some physicians lay considerable stress upon these factors. It is recommended by some that the carbohydrate and fatty constituents of the diet should be reduced in order to prevent an increase of the patient's weight. Malt liquors are excluded although light wines—such as Moselle—in moderate

quantity are permitted. The rationale given for this treatment is that by reason of the presence of osteophytes the joint is in an 'extremely irritable condition', and that any addition to the body weight increases this irritability. However, there is no doubt that this treatment is based upon erroneous pathological views. Sufferers from osteo arthritis are so apt to lose weight from the constant exhausting pain, that it seems desirable that the diet should be generous, strengthening, and digestible, and yet not of such a nature as to favour intestinal putrefaction.

With regard to medicine—gumacum, sulphur, arsenic, and the iodides all have advocates.

In conclusion, it is my pleasant duty to express my thanks to those—too numerous to mention in detail—who have assisted in various ways in this research. Certain acknowledgements have already been made in the text. In particular, the work owes very much to the help and criticism of Professor Shattoek, who honoured me by allowing the experimental work to be performed in his laboratory at St Thomas's Hospital, and whose kindness, sympathy, and encouragement have been stimulating and inspiring. To Sir Arthur Keith I am indebted for much help and for granting me permission to work at the Royal College of Surgeons. The Medical Research Council have generously defrayed the expenses of the research by a grant from their funds. My colleagues at the Seamen's Hospital, Greenwich, particularly Professor Hewlett and Dr Arthur Davies, have rendered valuable assistance. Finally, I must express my gratitude to the curators of many pathological museums and to Dr Strangeways for the loan of specimens, also to Dr Haward of the Ministry of Pensions, through whose help I have had special facilities for examining pensioners suffering from disabilities of the joints.

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PHARYNGEAL DIVERTICULUM AND ITS SURGICAL TREATMENT, WITH A RECORD OF TWO CASES.

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A POLCII-TIKI protrusion from the posterior wall of the lower part of the pharynx close to its junction with the œsophagus has been variously described as a 'pulsion diverticulum' of the œsophagus, 'pharyngocele', 'pharyngo-œsophageal diverticulum', and 'grenz' diverticulum. Though it is usually stated that Mondiere² was the first, in 1883, to describe the condition clearly, priority must certainly be claimed for Sir Charles Bell.³ The latter, in 1816, recorded a case of difficulty in swallowing which was temporarily relieved by the passage of œsophageal bougies. The patient died from other causes than that associated with the throat, and, at the post-mortem, a posteromedian pouch was discovered. This astute observer not only pointed out its pharyngeal origin, but also tendered the explanation which, in the light of future experience, has proved the most plausible one. He assumed the sequence of events to be difficulty in swallowing due to a spasmodic contraction of the sphincter at the lower part of the pharynx, hypertrophy and fasciculation of the pharyngeal musculature and finally herniation of the mucosa between hypertrophied muscle bundles of the inferior constrictor. He likened the condition to the hernial protrusion of mucosa which so frequently occurs in the hypertrophied bladder due to urethral obstruction. Credit is likewise due to Bell for being the first to suggest the possibility of alleviating such conditions, when associated with a swelling in the neck, by the establishment of a cervical fistula.

The next important contribution to the subject of pharyngeal and œsophageal pouches was that of Rokitsky,⁴ who classified such pouches according to whether they originated primarily from pressure from within or traction from without, and hence the terms 'pulsion' and 'traction' diverticula. It is to the former of these that the pharyngeal diverticulum belongs. In 1877, Zenker,⁵ in his classical study of 27 cases which came to post-mortem, laid a sure foundation for the symptomatology, diagnosis, and morbid anatomy of this condition. Though again subsequently overlooked he further established clearly the pharyngeal origin of such pouches. It is of interest to recall his prediction with regard to the operative possibilities of the condition: 'The radical cure of diverticula by surgical operation from without is at present one of our vain wishes, yet we are hopeful that even this operation conducted on Lister's plan may at some future day be performed without danger.'

The first recorded operation was that by Nicoladoni⁶ in 1876, when a fistula was established, but the patient died on the sixth day from pneumonia. In 1890 von Bergmann⁷ successfully excised a diverticulum with however the formation of a temporary fistula. Koehler⁸ in 1892 performed the operation with healing per primum. Since that time numerous cases submitted to operation have been recorded and of late years—probably owing to modern methods of diagnosis—the numbers recorded have rapidly increased. Deis⁹ has succeeded in collecting statistics of 149 operations. Butlin¹⁰ in ten years operated on 8 cases and in the Mayo Clinic¹¹ no fewer than 35 cases have been operated on.

Pathological Anatomy—Though it had been shown clearly by Bell and Zenker that the site of origin was pharyngeal and not œsophageal it was Killian¹² who first clearly established that the pouch is a protrusion of the mucous membrane between the transverse and oblique fibres of the cricopharyngeus muscle in the mid line posteriorly (*Fig. 92*), and this has been abundantly confirmed by Goldmann,¹³ Keith¹⁴ and others. As to the

pouchs increase in size, it descends and tends to be deflected to one side of the mid-line usually to the left. It passes down behind the œsophagus and carotid sheath and lies between the prevertebral and pretracheal layers of cervical fascia, and may eventually occupy the posterior mediastinum. The sac is usually pyriform in shape, and its pharyngeal orifice is as a rule moderately wide. The sagging down of the sac very soon brings the pharynx into alignment with it, the upper extremity of the œsophagus appearing as a narrow aperture on the anterior border of the neck of the sac. This readily explains



FIG. 92.—Pharyngeal pouch shown, the relation of the neck to the two portions of the cricopharyngeus muscle (Killian). (1) Uvula (2) Greater cornu of the hyoid bone (3) Oblique portion of cricopharyngeus muscle, (4) Diverticulum (5) Transverse portion of cricopharyngeus muscle (6) Esophagus (7) Trachea, (8) Recurrent laryngeal nerve, (9) Thyroid gland (10) Thyroid cartilage.

how all food and likewise how all instruments pass more readily into the diverticulum than into the œsophagus. The wall of the sac varies in thickness in different cases. So thin is it in some, that great care must be taken not to tear the wall (Bevan),²⁰ whilst in one of the cases here recorded, the wall when contracted measured 1 cm (Fig 97). Though variable in thickness the constituents of the wall are remarkably constant. It is lined by stratified squamous epithelium which in some cases shows hyperkeratosis, in others ulceration, and in a few this has gone on to malignant degeneration. In the submucous coat there may or may not be a muscularis mucosa, and this is in accord with the variability of the boundary of the œsophageal muscularis mucosa. Around the neck of the proximal part of the sac are usually found loose fasciculi of striated muscle, arising from the inferior pharyngeal constrictor. The outermost coat, or tunica propria is derived from the pharyngeal fascia and it is this coat which largely determines the thickness of the wall. The loose areolar tissue which separates the tunica propria from the lining membrane gives to the diverticulum when grasped between the fingers the sensation experienced when one grasps the stomach. Moreover, this loose intervening layer provides a ready line of cleavage, and permits of submucous excision.

Etiology—It may be stated at once that there is no evidence that the type of œsophageal diverticulum

here described is ever congenital in origin. True, certain lateral diverticula and sinuses are attributable to defective closure of branchial clefts.¹⁴ So far, however, no postero-median pouch has been met with in the new-born or in childhood. The majority of cases have occurred in male subjects past middle life, and, in a series of cases recorded by Stetton,¹⁵ the average age was 54. In 27 cases recorded by Zenker there was no female, and in the Mayo series of 35, the ratio of male to female was four to one.

The diverticulum is essentially a herniation of the mucosa through the musculature of the pharynx. One or both of two factors must operate—one, an abnormal increase of intrapharyngeal tension, the other some localized weakness in the posterior pharyngeal wall. A weakness in the posterior pharyngeal wall has been described at a point where the longitudinal fibres of the œsophagus diverge, the so called Luncer Hekermann¹⁶ triangle. But it has already been stated that the protrusion begins at a point between the oblique and transverse portions of the cricopharyngeus muscle. Though a congenital weakness at this point is conceivable, there is no proof of such existing. Probably far more important is the factor of prolonged abnormal intrapharyngeal tension. The second stage of normal deglutition though initiated by volition is chiefly an involuntary and complex reflex action. The oropharynx and nasopharynx are closed off, respiration is inhibited and the larynx is suddenly drawn upwards and forwards the bolus passing downwards and backwards towards the lower pharyngeal outlet. Killian from pharyngoscopy and anatomical observations demonstrated that the transverse fibres of the

œsophageus act is a sphincter to the upper end of the œsophagus, and Goldmann¹² has confirmed this by interesting observations on two patients during an operation for goitre. The pharynx and œsophagus presented themselves as moderately-filled air sacs, and were separated by a ring constriction which lay at the level of the lower half of the cricoid. When the patient swallowed, the saliva was seen to bound against the constriction, and, after a short time, the ring opened, and the pharynx and œsophagus became continuous. Immediately thereafter the constriction formed again. Recently, we have had a convincing proof of the sphincteric action of the lower part of the pharynx in a case of suicidal cut-throat. The larynx had been completely divided immediately above the true vocal cords, and only the posterior wall of the pharynx remained intact. A finger passed down the pharynx encountered a conical contracted sphincter at the level of the lower half of the cricoid cartilage. The patient was asked to swallow. The muscles on the posterior pharyngeal wall contracted, partially elevating the larynx, and, almost simultaneously the sphincter relaxed. The opening of the sphincter was sudden, and, after a brief interval of from half to one second, slowly closed again. It is easy to conceive that incoördinate relaxation of this sphincter would considerably increase the intra-pharyngeal tension and that the strain would be maximum at the point at which the bolus is arrested. In the two cases observed by us, a definite difficulty in swallowing had been noted, in one case for fifteen years and in the other ever since boyhood, and such cases are by no means exceptional. Further, there are cases on record in which there was noted a definite organic stenosis^{17, 18} at the upper œsophageal extremity, some of congenital origin some fibrotic, and it is noteworthy that the examples of diverticuli in early life have been associated with such stenoses.⁶ Trauma has been cited as the predisposing cause in a few cases, but its etiological relationship is difficult to establish and must be regarded as exceptional.

Once the protrusion has begun, the displacement forwards of the upper aperture of the œsophagus renders still more difficult the passage of food into the œsophagus, and the propulsive force of the pharynx is expended in dilating the pouch. In accord with this is the phenomenon so consistently noted, that nothing enters the œsophagus until the pouch is filled. Radioscopic examination in one of our cases confirmed the observation of Hartmann¹⁹ that food distends the neck of the filled sac and then overflows into the œsophagus.

From the foregoing anatomical, physiological, and clinical observations it may be concluded that the prime etiological factor is some interference with the outlet of the pharynx. Whereas in exceptional cases there may be some readily recognizable organic stricture in the majority of cases it is probably of a functional nature and due to an incoördinate action between the propulsive and sphincteric elements of the neuromuscular apparatus.

Clinical Manifestations—In a number of cases there had been a history of some defect in swallowing for many years before the typical symptoms attributable to a pouch were noted. In one of our cases that difficulty dated back to boyhood and was the only symptom manifested for thirty-seven years. It is quite possible that in the majority of cases the earliest symptoms are due not to the pouch but to the deranged neuromuscular mechanism of deglutition.

When a pouch has formed the most characteristic symptom is the regurgitation at variable periods after meals of unaltered food. At first the quantity may be small but it tends progressively to increase. Associated with this there are often gurgling noises which are a source of annoyance to the patient and occasionally may be audible at a considerable distance. A noticeable symptom is an accumulation of saliva in the pharynx and in certain cases rest at night may thereby be disturbed. The initial difficulty in swallowing becomes more pronounced the sac enlarges and in an extreme case such as the first one here recorded the partaking of a meal is looked forward to with anxiety and even dread. Before anything passes down the œsophagus the patient must first fill his pouch and any misadventure may cause regurgitation of its contents. The patient then experiences a choking sensation bends forwards and empties the pouch and the

ordeal has to be repeated. Only by exercising great caution when the pouch has been filled can the patient succeed in swallowing sufficient nourishment to maintain nutrition.

In moderate-sized pouches the swelling may be visible on one or other side of the neck, more often on the left side. Each swallow of the patient causes the swelling to increase in size, and the larynx and trachea may be displaced and the sternomastoid bulged forwards. When the sac is full, pressure applied to it may empty it, particularly if the patient be lying on his side. The rate of increase in symptoms varies, but on the average in a series of 35 cases¹¹ the time from the onset of the symptoms to the patient's undergoing operation was five and a half years.

Diagnosis—In a well-marked case, the diagnosis can usually be made from clinical manifestations alone, and can be readily confirmed by radioscopic examination. In the early stage, various conditions may be confused with it, such as simple or malignant stricture of the œsophagus, and cardiospasm. In all of these there may be difficulty in swallowing, excessive salivation, and arrest of bougies, but the true nature of the malady can always be revealed by resort to radiography. The latter has now replaced not only the use of the bougies but even œsophagoscopy, and is moreover a much safer procedure.

Complications and Terminations if Untreated—The progressive difficulty in swallowing eventually leads to inanition, and in numerous cases death from starvation has resulted. Stagnation of contents may give rise to ulceration of the mucous lining, and may cause secondary suppuration in and around the wall of the sac, or lead to perforation. Intercurrent lung complications are not infrequently the cause of death. Carcinomatous degeneration has been recorded in several cases.

Treatment—Hitherto, attention has been almost entirely directed to the removal of the sac. Sight must not be lost, however, of the possibility that in the sac we are dealing with merely a secondary condition, and to ensure a radical cure an effort must be made to overcome the primary cause. Thus, if an organic stenosis of the œsophagus be present one would naturally first dilate the stricture. This principle is equally applicable to cases in which the obstruction appears to be functional in character. In very early cases, Bevan¹⁰ recommends the passage of bougies, with the object of opening up the pouch. It is not unlikely, however, that the good resulting from this measure is due to dilatation of the pharyngeal sphincter. In very early cases it is conceivable that such measures may be the means of arresting the development of the sac. In late cases such dilatation should supplement the extirpation of the sac.

Removal of the sac, despite the recorded series of successful operations, is nevertheless to be regarded as a procedure fraught with danger. The patient is usually of advanced years and is frequently debilitated. The sac of necessity contains infective organisms, communicates with a septic channel, and lies in a cellular plane ill-fitted to deal with infection and continuous with an inaccessible space—the posterior mediastinum. The attendant risk is evidenced by the numerous operative methods that have been devised.

To combat the state of inanition, a primary gastrostomy has been strongly advocated by some surgeons, and is undoubtedly advantageous in late cases. To minimize infection from the interior of the sac, careful attention must be paid to the teeth and gums, and lavage of the sac with a mild antiseptic is a useful pre-operative practice. An empty sac is an essential for a safe operation, and measures must be taken to ensure this by posture, and by pressure over the sac immediately after the lavage and prior to the operation. Cases have been recorded in which this was not attended to, and in which aspiration of infected material led to pulmonary complications.

In regard to anaesthesia, Lupke¹ and Bevan¹⁰ strongly recommend local and regional anaesthesia as precluding the risks of aspiration and avoiding post-operative sickness. On the other hand, the majority of successful operations have been carried out under general anaesthesia, and when such anaesthesia is employed, the intratracheal insufflation of ether will probably be the one of choice.

As to the methods of operation, these have been diverse, such as simple pharyngopexy, magnum of the sac, the two-stage removal, and—what would appear to be the

ideal operation—the one-stage radical extirpation of the sac. Pharyngopexy advocated by Hill²² will probably be reserved for small pouches in old and debilitated subjects. Invagination is likewise only applicable when the sac is small. In the larger sacs, as pointed out by its introducer Bryan²⁰ it is not free from danger, since the invaginated sac may be displaced upwards and occlude the pharyngeal orifice.

A two stage removal of the sac was first practised by Goldmann with a view to minimizing the risk of cellulitis in the planes of the neck, and this, with numerous modifications, has been widely adopted. Goldmann's²² operation consists in freeing and bringing out the sac ligating its neck with silk and gently packing around, and removing the sac at the second operation. Deis⁹ carries out the same procedure, but leaves the sac to slough off of its own accord. In Mayo's¹¹ method, the sac is freed and the skin sutured down to its neck, and at a subsequent operation the sac is excised. Another modification is to free the sac twist it at its neck and gently pack around, when the fascial planes are all well off, the sac is removed and its pedicle sutured.

The method adopted in the two cases here recorded was to offer still greater protection against the danger of cellulitis. It consists essentially in freeing and bringing out the sac at the first operation and in a submucous excision of the lining at the second, the tumour proper being left adherent to the skin, and the planes of the neck being left undisturbed.

In the one stage operation the sac is isolated and removed and the neck is variously dealt with. Koehler's method is really a 'cuff' operation, and is very similar to that of dealing with an appendix stump. Perthes²¹ divided the neck between clamps, and dealt with the stump after the Movnik method of closing the duodenum in a pylorotomy, the suture-line being vertical. Lexer conserved a pedicle which he invaginated so as to form a valve like internal protrusion. It is necessary to utter a word of warning about division of the neck. Unless great care be taken traction on the sac may pull out a portion of the normal pharyngeal wall which may be mistaken for the neck of the sac. In a case recorded by Downes,²³ all save a narrow strip on the right lateral wall of the upper end of the oesophagus was inadvertently removed with the sac, and it was found necessary to excise the remaining portion and to do an end-to-end suture.

The one stage operation makes an obvious appeal to the surgical instinct, and in favourable cases has much to commend it. Discretion, however, will often dictate the more tedious but surely the safer course of a two-stage operation and it must be left to the judgement of the surgeon in the individual case to decide which is the method of choice.

Common to all the methods previously discussed is the mode of approach. The usual incision is along the anterior border of the sternomastoid. Occasionally, however, a collar incision such as is used in thyroidectomy is employed. At the anterior border of the sternomastoid the deep fascia is incised and the muscle retracted laterally. The omohyoid is freed and displaced outwards or divided. The carotid sheath is exposed, and, after dividing the middle thyroid vein is retracted laterally. The thyroid gland is displaced medially and at this stage the diverticulum will be visible in the depths of the wound. Crossing it is the inferior thyroid artery and if this impedes access it may be divided. The lateral expansion of the pretracheal layer of fascia is divided, and the sac may now be delivered. As a rule the pouch lies amid loose cellular tissue and can be readily freed, but in exceptional cases preceding inflammatory changes in and around it will may render this stage of the operation exceedingly difficult.

Post-operative Course and Treatment.—The complication most to be feared is a spreading infection of the cellular planes of the neck with its sequelæ mediastinitis. As has been pointed out this may be effectively prevented by doing the operation in two stages, and particularly by the method of submucous excision. In the one-stage operation infection may arise from continuation of the wound at operation or more frequently, from subsequent leakage. To avert the latter the indications are (1) To give rest to the wounded pharynx as far as possible, and to render as aseptic as possible the secretions of the mouth. For several days oral feeding is better avoided, glucose enemata being

administered by the rectum. Feeding through an œsophageal tube passed at the time of operation has been practised, but is no longer recommended. (2) Free drainage must be established, so that any leakage may find ready exit.

A temporary fistula is to be expected in about 50 per cent of the cases. Recently, however, some operators have recorded short series of cases with a much higher percentage of healing per primam. With few exceptions, the fistulae close within a few weeks, and are of little moment if they do not occur prior to the sealing off of the cellular planes by granulation tissue.

ILLUSTRATIVE CASES

Case 1—Mr. A. H., age 53. Patient states: "I can remember quite clearly that, when a boy about 9 years of age, I began to realize there was something not quite right in regard to my swallowing food. When any lumps or knots of oatmeal were present in my porridge to swallow them without chewing was both difficult and disagreeable. Throughout early manhood I could never eat dry bread except very slowly and with a great deal of chewing, and I developed the habit of taking a fair quantity of liquid with my meals. I could always manage to obtain sufficient nourishment, provided I had plenty of time. Further, in drinking fluids such as milk or water, it had to be taken in small quantities and not drunk continuously."



FIG. 93.—Case 1. With the sac empty.



FIG. 94.—Case 1. Sac distended with food.

"It would be about seven years ago that I first began to realize there was something radically wrong. I had a bad taste in my mouth in the morning and coughed up small quantities of substance from my throat. Six years ago I found that, hours after meals, food would come into my mouth as fresh as when I had taken it first. Further, especially at night when in bed, I was surprised by hearing gurglings in my throat. Sometimes these sounds were quite pronounced."

"Three years ago, I sought advice, and radiograms were taken and a pouch was recognized. Soon thereafter I developed pneumonia. Following the attack I discovered that I could empty the pouch by simply holding my head down over a basin. This allowed me to carry on with comparative comfort, but, during the last three years, swallowing has become more and more difficult. Formerly I weighed 13 stone, now I weigh 11 stone 13 lb."

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PHYSICAL EXAMINATION, Jan 12, 1920—Patient was fairly healthy in appearance, though somewhat emaciated. There was nothing to be made out on simple inspection of the neck (Fig 93). Patient was given a glass of water. This he could only swallow in mouthfuls at a time. A swelling began to appear on the left side of the neck, and this grew larger with each swallow. The swelling occupied the lower carotid region and bulged into the subclavian triangle. Apparently the whole of the tumblerful had gone directly into the pouch. The water he readily brought back by holding his head on one side and pressing on the swelling in the neck. It was found that he could swallow air into the pouch if he held his nostrils. Observations were made while the patient took breakfast. His endeavour was first to fill the pouch. He took 10–15 ounces of milk with his porridge, and, with each gulp, the swelling in the left side of the neck became more prominent, until it eventually assumed the dimensions shown in Fig 94. The lower half of the sternomastoid was bulged forwards and laterally, and the larynx and trachea were displaced a little to the right. When the sac was full, the patient began to have some difficulty in speaking, and he breathed with

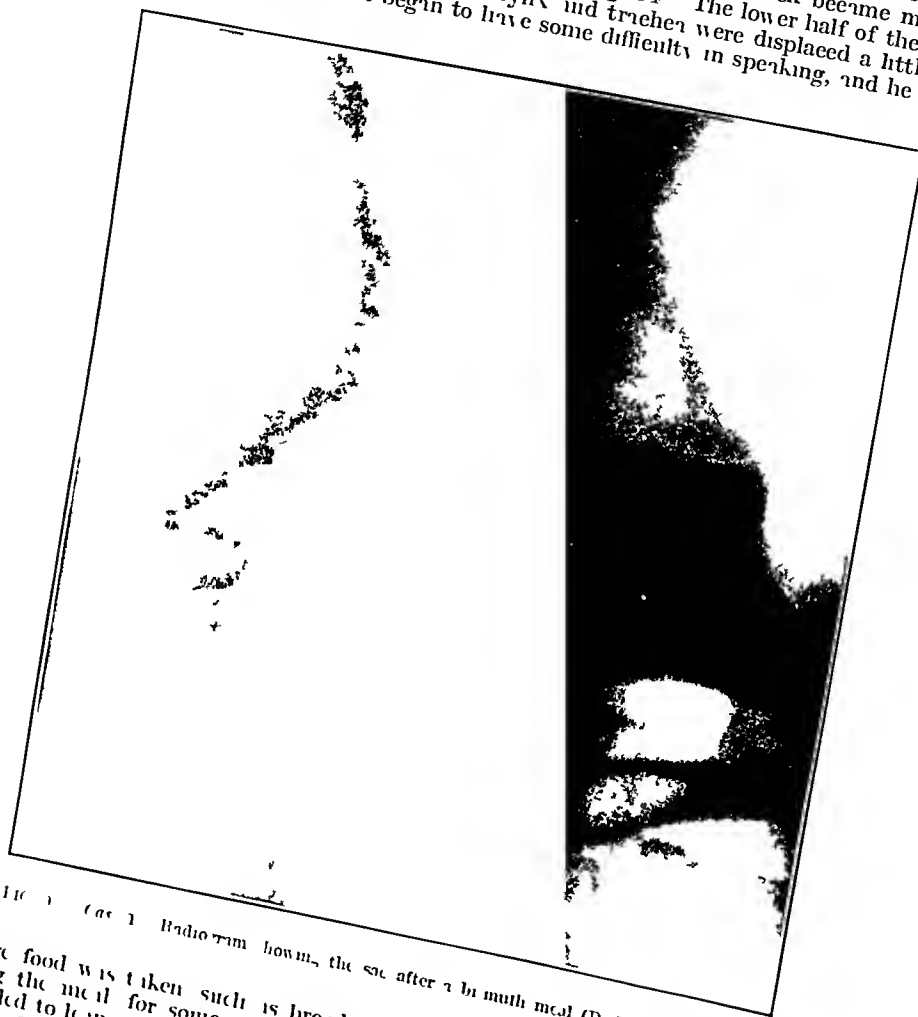


FIG. 94. (a) Radio-gram showing the sac after a bismuth meal (D. W. Hope Fowler)

caution. More food was taken such as bread tea, and also a little bacon, and finally Benger's Food. During the meal for some unknown reason, a little regurgitation caused irritation, and he was compelled to lean well forward on to one side over a basin which he always had at his feet, and the whole of the contents of the sac were disgorged. He thereafter had to begin again to fill the pouch before he could get anything to pass down the oesophagus. A radiogram was taken after a bismuth meal (Fig 95). The pouch will be seen to extend down into the mediastinum. The upper part of the sac is full of air and the sternomastoid is seen bulging laterally.

OPERATION. 1st stage Jan 17.—Sir Harold J. Stiles operated, chloroform and ether being employed, and while the patient was under anaesthesia an oesophagoscope was carried out by Dr Logan Turner. The oesophagoscope was found to pass directly into the sac and one could see the rugose wall of the diverticulum expanding and contracting with the respiratory movements. On retracting the oesophagoscope the orifice was observed as a vertical slit just to the right of the mid-line on the posterior wall of the lower part of the pharynx. The patient was placed in the supine position

with the head slightly turned to the right side. Incision along the interior border of the sternomastoid from the inner end of the clavicle to about the level of the hyoid bone, from a point at the junction of the middle and lower thirds of this incision a second short incision was carried downwards and outwards. The external jugular vein was divided between clamps. The anterior border of the sternomastoid was defined and retracted outwards. The omohyoid muscle was freed and retracted upwards and outwards. The middle thyroid vein was divided between clamps and the carotid sheath retracted laterally. Deep in the interval between the carotid sheath and the trachea the œsophagus could be seen and palpated, and behind this, extending outwards behind the carotid sheath and downwards behind the œsophagus into the thorax, was a thick walled pouch.

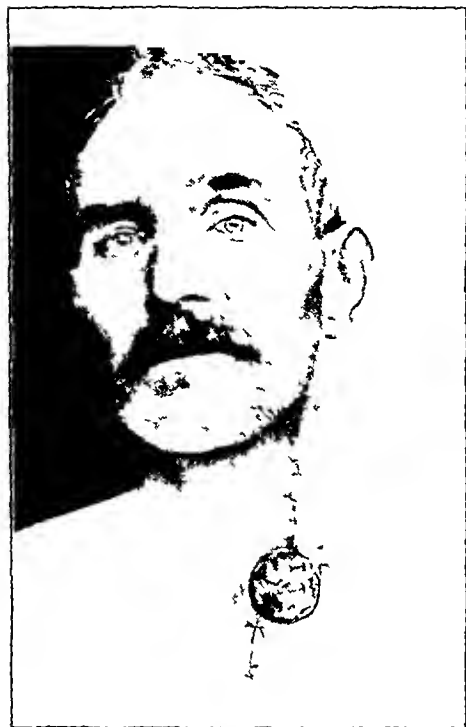


FIG. 96.—Case 1. After first stage of the operation.

The depressor muscles of the hyoid were freed a little more and retracted well inward with the larynx and trachea, and after tearing through the thin lateral expansion of the peritracheal layer of fascia, the sac was gripped and dislodged from the mediastinum, a few loose fibrous connections being divided with the scissors. The neck of the pouch was defined and was found not to extend much above the level of the cricoid cartilage. The wall of the sac was thick and was comparable to that of the stomach. The fundus of the sac was brought out of the wound between the carotid sheath and the thyroid, and in front of the interior border of the sternomastoid. The wound was closed about the sac, the skin margins being fixed to the body of the sac (Fig. 96).

2nd Stage, Jan. 29.—Chloroform and ether given. The base of the projecting portion of the sac was incised until the loose submucous tissue plane was reached. By blunt dissection the mucosa was separated off for about two inches from the skin surface and the fundus, together with the freed mucous lining, was resected. Fig. 97 shows a section of the portion removed. The edges of the mucosa were picked up with catch forceps, and the finger on being inserted, encountered the pos-

terior aspect of the cricoid cartilage. On account of the depth, the finger could not be inserted down to the œsophagus. One could just detect the space between the orifice of the pouch and the opening of the œsophagus.

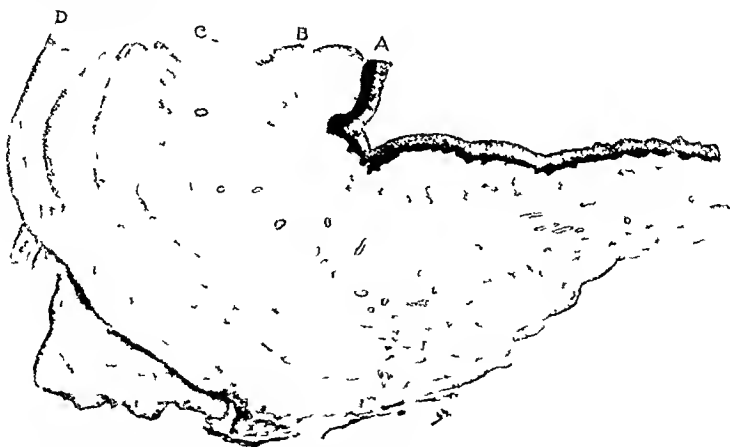


FIG. 97.—Case 1. Portion of fundus of sac removed at second stage operation (Micro section $\times 75$). (A) Lining squamous epithelium, (B) Submucous layer, (C) Tunica propria, (D) Granulation tissue and blood clot.

The free edge of the mucosa was inverted by a roll-in suture of chromicized catgut, and further invaginated by interrupted sutures. The stump was then allowed to retract into the

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deeper parts of the wound. A little iodoform bismuth paste was smeared into the sinus and the orifice was partially closed by means of skin sutures. It is to be noted, therefore, that the operation was carried out within the submucous layer of the diverticulum and the planes of the neck were not opened. Fig 98 shows the condition after this stage of the operation.

POST-OPERATIVE TREATMENT—After the first stage operation, glucose sahne enemata were administered for two or three days, and thereafter fluid nourishment was given by the mouth following the second stage operation, glucose enemata were again administered, and on the fourth day small quantities of fluid nourishment were allowed by the mouth.

The patient's condition remained excellent after both operations. On the eighth day after the second operation a little fluid food crept through the wound in the neck. This continued for eight or nine days and then ceased. Two and a half weeks after the second operation the patient was allowed to go home with the wound in the neck almost closed.

The following extract from letters received from the patient shows the progress of the case—

May 12, 1920. My appetite is good, and I am feeding up freely on porridge, eggs, and pudding. I can take food with little discomfort, and the wound has healed up clean and well. I have put on weight rapidly. There has always, however, been a quantity of food lodging in the throat, which I have discharged from the mouth after each meal. The amount began with about two tablespoonfuls but is now about three tablespoonfuls. I find I cannot swallow mustered scones or meat readily and food seems to go down in a jerky manner.

March 21, 1921. My weight is now over 12 stone. My throat condition has not troubled me so much during the last four months. The quantity retained in my throat after meals does not seem to be getting any larger, and I can swallow with a little less trouble. I may say that there is nothing I cannot take provided I get time and have a fair quantity of fluid with my meals.

April 4, 1922. I can manage with ease to cut and swallow practically my ordinary material. Butcher's meat potatoes, and anything of a pasty nature seem to clog the passage, and when trying to force matters a little bit, I find the food gets into a pocket and there is a prominence which can be felt by the hand on the left side of the neck. The amount of food which lodges in the throat is quite a breakfast cupful. During the last six months however I am inclined to think it has not got any worse and there is it times some feeling as if the condition was growing more comfortable. My weight to day is 12 stone 5 lb.

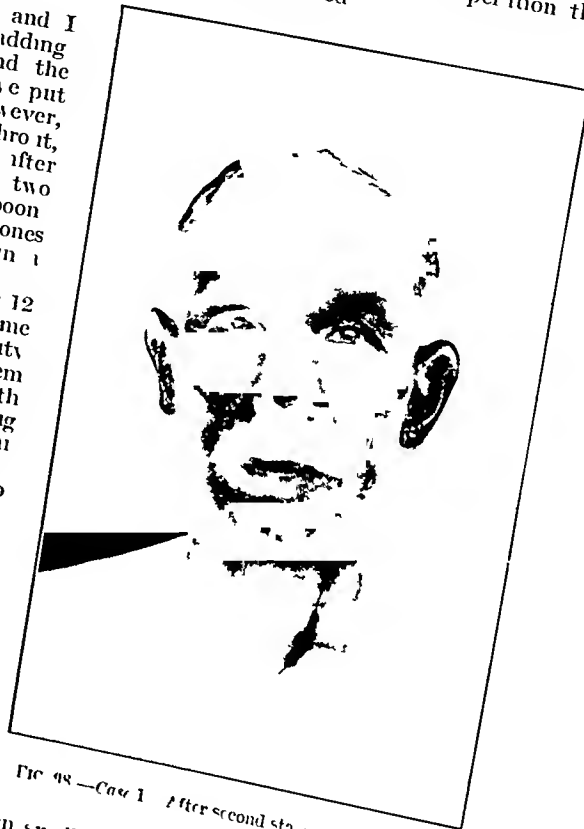


FIG 98.—Case 1. After second stage of the operation

Case 2. Miss G. age 67. Complaints of difficulty in swallowing, clicking noise in throat, and fullness of the neck after eating solid food. For over twenty years patient had been conscious of a difficulty in swallowing and since its appearance swallowing became progressively more difficult. This she repeatedly noticed herself on a clicking noise which she made just after swallowing. Two years ago she began to have vomiting after meals and she noticed that what she brought up always consisted of the first part of the meal taken. Her medical man suggested the possibility of an oesophageal pouch by day and during the night. She was also now considerably troubled by bringing up mucus both level of the cricoid cartilage and a diagnosis of a congenital and soft solid diet and therefore patient weighed 6 stone 4 lb. She was put on a generous fluid and soft solid diet and thereafter steadily put on weight until at the present time she weighs 8 stone. The vomiting up of the first part of the meal however has continued and the constant bringing up of mucus has become more pronounced interfering greatly with her rest at night. She has latterly been more conscious of an uncomfortable fullness on the right side of the neck after eating and this is only relieved by vomiting.

ON EXAMINATION.—Patient is a thin and delicate looking old lady. A slight fullness of the neck is noticeable under the lower half of the right sternomastoid muscle. On palpation a deep seated soft swelling can be made out to the right of the trachea. Pressure on this gives a curious dull crepitant sensation.

X-RAY SCREEN EXAMINATION—When patient swallows bismuth it is seen to enter a pouch lying to the right of the mid line and extending down to the manubrium sterni, the shadow being rather larger than a hen's egg (*Fig 99*). When this pouch is filled, but not till then, the bismuth is seen to pass down the œsophagus to the left of the pouch. After the bismuth meal is all swallowed the shadow of the filled pouch remains.

DIAGNOSIS—Pharyngeal diverticulum lying to the right of the œsophagus. As the condition was causing great interference with the patient's comfort, and especially with her rest at night, operation was recommended. In view of her age and somewhat frail constitution, a two stage operation was decided on.

OPERATION, 1st Stage, Dec 6, 1921 (Mr Wilkie, assisted by Dr T W E Ross)—To ensure that the diverticulum was relatively clean and empty, the patient was made to swallow weak boracic lotion, and then to bend over and compress the right side of the neck, thus washing

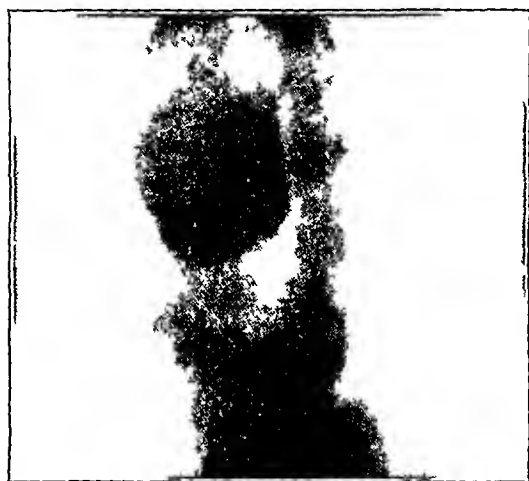


Fig 99—Case 2. Radiogram after bismuth meal.
(Dr T W E Ross)

out the pouch. Under chloroform an incision was made along the anterior border of the lower half of the right sternomastoid. On dividing the deep fascia the omohyoid muscle was exposed and divided. The pretracheal layer of deep cervical fascia was divided, and the pouch was exposed, lying under cover of the thyroid gland and medial to the carotid sheath. The inferior thyroid artery was visible, crossing on the anterior aspect of the pouch, it was not divided. The pouch was readily freed except at its apex, which lay just behind the episternal notch. On dividing one or two fibrous adhesions at this point it was readily brought out into the wound. It measured three and a half inches in length, and had a broad neck which rose from the posterior pharyngeal wall. The œsophagus lay directly in front of the proximal part of the sac and a few longitudinal muscular fibres from the inferior constrictor extended on to its neck. A few horizontal muscular fibres from the pharynx were also seen on the proximal half inch. On picking up the sac between the finger and thumb the feeling experienced was almost identical with that on palpating the urinary bladder. On pull-

ing forward the sac and examining it from behind, it appeared to be continuous with the pharynx and it was difficult to detect where the sac ended and the pharynx began. The pouch was brought out at the upper end of the wound. Three linen sutures were passed through its fibrous coat near the neck, and these were left long and brought out through the wound. Two catgut sutures fixed its fibrous sheath to the depressor and sternomastoid muscles. Some ribbon iodoform gauze was packed into the space from which the sac had been removed, and a small strip into the retropharyngeal space above it. The wound was closed with silkworm gut sutures, leaving the sac protruding from its upper end.

The patient was given rectal salines for the first twenty four hours and thereafter food by the mouth. On the second day she could swallow solid food without difficulty. Five days after operation the gauze packing was removed.

2nd Stage—Eleven days after the first operation, under chloroform ether anaesthesia, an incision was made at the level of the skin through the tumor proper into the submucous tissue. It was found that the mucous sac could then be separated readily down to its junction with the pharynx. Here it was caught with peritoneal forceps and the sac cut away. A finger was then introduced through the neck of the sac into the pharynx. The opening of the œsophagus was felt as a narrow transverse slit on the anterior wall of the neck. The forefinger was introduced into it with difficulty and slowly dilated it. It was then seen that the whole of the mucous lining of the neck of the sac had not been removed, and another ring three sixteenths of an inch broad, of mucous membrane, was taken away. The cut edges were then enfolded with a roll over suture of 00 twenty day tanned catgut. Before this suture was completed a large sized olive headed œsophageal bougie was passed by the mouth, and this showed that the pharynx had not been unduly constricted. It was clearly demonstrated that just below the suture line the bougie was arrested and considerable pressure was required before it passed onwards into the œsophagus, and further, that on withdrawing it, it was arrested at the same point. This manoeuvre was repeated and the observation confirmed. One catgut suture was put in, drawing the walls of the sheath together. Throughout the whole operation the fibrous sheath of the diverticulum remained adherent to the skin.

The microscopic appearances of the part removed are shown in *Fig 100*.

POST OPERATIVE COURSE—For three days glucose enemata were given at four-hourly intervals, nothing being taken by the mouth. The mouth was washed out every two hours with phenol sodique, and the patient sucked formamin tablets on her own suggestion. On the fourth day she was given sterile water to swallow and on the fifth day clear soup. Thereafter a more generous diet of various sterilized liquid foods was given. On the eighth day a small fistula developed and remained open for five weeks, when it finally closed. Very little ever came through the fistula even when liquids were swallowed. There was no cellulitis of the neck or other complications. The patient went home six weeks after the second operation with the wound healed, and could swallow ordinary solid food without difficulty.



FIG. 100.—Case 2. Micro-photograph of portion of pouch removed at the operation.

The points of special interest in this case are the right-sided position of the diverticulum, and the determination during the second operation that the upper œsophageal sphincter was narrowed and offered a definite resistance when a finger and a large bougie passed through it. The latter procedure it was hoped, and with some reason would have a beneficial therapeutic effect in facilitating swallowing and preventing undue intra-pharyngeal pressure.

CONCLUSIONS

1 A pharyngeal diverticulum is an abnormal protrusion of the mucous membrane of the lower part of the posterior wall of the pharynx, between the oblique and transverse fibres of the cricopharyngeus muscle.

2 It occurs most frequently in men past middle life.

3 The condition is more common than has hitherto been supposed, for, although only some 200 cases have been recorded, the majority of these have been within comparatively recent years.

4 Two etiological factors are involved—the one—a weakness of the wall—is problematical, the other is increased intrapharyngeal pressure.

5 In exceptional cases, an organic stenosis has been present—in the majority the primary cause would appear to be the inco-ordinate action between the propulsive and sphincteric elements of the pharyngeal muscle.

6 Radioscopy has replaced all other special diagnostic methods.

7 Treatment must be directed to both cause and effect by dilatation of the stenosis, be it organic or functional, and extirpation of the sac.

8 The one stage operation is ideal, but not devoid of danger the chief risk being leakage and cellulitis of the planes of the neck

9 The two stage operation is that recommended in feeble and elderly patients

10 The modification of the two stage operation, in which a submucous excision of the sac is made at the second stage, though perhaps not the most radical, is however, the one involving least risk to the patient's life

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THE REMOTE EFFECTS OF GUNSHOT WOUNDS OF THE HEAD.

By L. BATHIE RAWLING, London

SYNOPSIS

- I —INTRODUCTORY
- II —SCALP WOUNDS
- III —NON-PENETRATING WOUNDS
- IV —PENETRATING WOUNDS —
 - a WITH HERNIA CEREBRI
 - b WITH FOREIGN BODIES RETAINED IN THE BRAIN
- V —PERICRANIAL WOUNDS
- VI —FRACTURED BASE
- VII —GENERAL REMARKS ON THE REMOTE EFFECTS OF HEAD WOUNDS, WITH
SPECIAL REFERENCE TO EPILEPSY, FITS, ETC
- VIII —DI COMPRESSION, WITH DETAILS OF 40 CASES
- IX —CLOSURE OR PROTECTION OF APERTURES IN THE SKULL
- X —POSTSCRIPTUM

I INTRODUCTORY

SUFFICIENT time has now elapsed since the Great War to justify expression of opinion as to the remote effects of gunshot wounds of the head, and other head-injuries of warfare. I have, at any rate, some justification for so doing, for, from 1914–20, I was in touch with this class of case (1st London General 1914–16, 34th (Welsh) General, India, 1916–18, 1st and 4th London Generals 1918–20, and from 1920 onwards, at the Ministry of Pensions Hospital, Ruskin Park).

Although service in India brought to my experience some new and interesting facts connected with gunshot wounds of the head, and malaria and heat-stroke cases with head complications, it was during the first six months of 1918 that I encountered the great mass of material which forms the basis of this paper. During that time I was in charge of 150 beds reserved for head cases at the 1st London General Hospital, together with another 100 at a convalescent home to which recovering cases were sent, and these 250 beds were usually occupied by head cases of all sorts and of all grades of severity.

Stress of work, foreign service and the general conditions prevailing, prevented me during the war from investigating the cases in the most desirable manner, but I have kept in touch with a considerable number of the men, many of whom write to me from time to time, and some of whom I see periodically. This paper is based on these cases, supplemented by observations carried out since, both at St Bartholomew's and at the Pensions Hospital. At the latter hospital I am in constant touch with patients suffering from head injuries and the investigation of these cases, in the comparative leisure of peace-time, has enabled me to curb enthusiasm, to modify earlier ideas, and to concentrate on the more practical points.

During the winter of 1920 and onwards, being in possession of details of 1000 cases, I was desirous of obtaining facts from that number but some had to be eliminated, 750 remaining. To these a form was despatched, requesting full information as to present condition, working capacity, etc. Replies were received from 452, from all parts of the world—the United Kingdom, ranch and town in Canada, Australia, New Zealand, Africa, and elsewhere.

The results here tabulated may, therefore, be regarded as representing the conclusions to which I have come after no inconsiderable experience. I would add also that, to avoid any bias, I have not read the Report on Head Injuries, issued by the Research Society, preferring to come to my own conclusions, irrespective of the opinions of those distinguished neurologists and surgeons who, sitting together, issued that report. If my ideas differ from theirs, time will show which is the correct view.

The injuries in these 452 cases represented —

	Cases
Scalp-wounds	17
Non penetrating wounds	121
Penetrating wounds	207
Perforating wounds	21
Fractured bone	16
Decompression	40
	<hr/>
Total	452

II SCALP-WOUNDS

Scalp-wounds are usually regarded as of trivial nature—the bone is unbroken and therefore the injury is of no serious import. Indeed in comparison with the frequent gunshot wounds of the head so frequently seen, the deduction seems fairly obvious. There is, however, another side to the picture, and the reports received from 47 cases of scalp-wound, in their life history up to six years subsequent to the reception of the injury, show that there is another aspect in the consideration of scalp wounds.

In most of these 47 cases the injury was incurred during a blowing-up process the patient often being ignorant as to what really happened. In the remainder, glancing shell-fragments had usually produced the wound.

An examination of record cards and close scrutiny of the history clearly show that in 22 cases (47 per cent) the injury was followed by a phase or syndrome which could be explained by no other hypothesis than that the patient was suffering from symptoms clearly pointing to definite contusion or laceration of the brain, or to intracranial hemorrhage. The facts in these 22 cases which give rise to this statement are as follows. In 12 cases the injury was followed by a period of constant and severe headache, often of so definite a character as to be the outstanding 'memory' of the case the one thing that the patient remembers after all these years, and of which some are in dread of recurrence. These headaches were unquestionably due to a general increase of intracranial pressure secondary to intracranial blood extravasation, with secondary oedema of the brain. This is confirmed by the statement in many record cards that lumbar puncture, frequently repeated, was carried out for the relief of the headache in many cases with the withdrawal of blood-stained cerebrospinal fluid. In 6 cases the injury was followed by fits of the Jacksonian type, by localized paralyses sensory disturbances, aphasia, etc., and in 5 of these cases the fits or paralyses persist at the present time. It is obvious therefore that the scalp-wound in these cases was complicated by a localized cerebral laceration or hemorrhage. In 4 cases the injury was followed by a prolonged period of unconsciousness accompanied by slowing of the pulse-rate, raising of the blood-pressure vomiting, etc., all symptoms indicative of intracranial blood extravasation, with generalized oedema of the brain.

These 22 cases, all of which were x-rayed, with negative findings in respect to osseous injury may be accepted as proof of the contention that intracranial complications were present, the lesion being in each case of such a nature as to produce definite clinical signs and symptoms.

In the remaining 25 cases the reports were not so clear, but in view of the significant facts elucidated in the 22 cases, it is obvious that the after-history of these scalp wound cases should be interesting perhaps also offering a guide to the early treatment of such wounds in the future.

THE REMOTE EFFECTS IN 17 CASES OF SCALP-WOUND

Do you suffer from	Headaches?	Yes 18 (severe 23, slight 15)	No 9
'	Insomnia?	Yes, 17	No 30
'	Giddiness?	Yes, 31	No, 16
'	Nervousness?	Yes, 25	No, 22
'	Any form of paralysis?	Yes 7	No, 40
'	Fits?	Yes 5	No, 42
Are you improving?		Yes 29 (60 per cent)	
Are you stationary?		Yes 16 (34 per cent)	
Are you getting worse?		Yes 2 (4 per cent)	
Are you at work	what work and hours?—		
	No work	5 (10 per cent)	
	At work	42 (light 19 per cent ordinary 70 per cent)	

It seems to me that the outstanding feature in these cases is this—in spite of the fact that 81 per cent complain of headaches 66 per cent of giddiness 53 per cent of nervousness 15 per cent of paralysis, 10 per cent of fits yet 89 per cent are working. It must be concluded I think, looking at the question from the broad point of view of present-day psychology that the symptoms of which the patients complain apart from fits and paralysis must be of the minor type.

Nevertheless there remain the 10 per cent incapacitated from work by reason of fits and palsies, and by severe and persistent headaches. It is probable that some of these would have benefited by early active treatment. Anyhow it is about time that the expression only a scalp wound was forgotten and that every case was treated on its own merits—with a clear basic understanding that the great majority of scalp-wounds of war time are associated with some degree of concussion of the cerebral change amounting in many cases to cerebral laceration and contusion, intracranial hemorrhages, etc.

Every case therefore demands the most careful investigation with special reference to the prolongation of unconsciousness, pulse rate, blood-pressure, persistence of headache, etc. Apart from benefit that might accrue by early operative treatment in selected cases, all cases require prolonged rest and convalescence, and many of these patients must be regarded as totally unfit for further active service.

In confirmation of these statements I would draw attention to a paper in *Brain* vol. xli by Geoffrey Jefferson, on the neurological findings in 54 cases of scalp-wound. These cases were seen and treated at a Base Hospital in France and in only five were no such evidences present. There were eleven definite local contusions of the motor cortex.

Jefferson's statements as to the earlier conditions and my findings as to remote effects are significant.

III NON-PENETRATING WOUNDS DURA MATER NOT PENETRATED

This section refers to various fractures of the skull, many complicated by the presence of hemorrhages and brain injury, but all having an intact dura mater.

ANSWERS WERE RECEIVED FROM 119 CASES

Headaches	Yes 110 = 92 per cent,	No, 9
Insomnia	Yes 59 = 50	No, 60
Giddiness	Yes 92 = 77	No, 27
Nerves	Yes, 85 = 71	No, 34
Paralysis	Yes 24 = 20	(Hemiplegia diplegia sup long sinus syndrome, aphasia, sensory disturbances, etc.)
	No 95	No 103
Fits	Yes 16 = 13 per cent	No 103
Present condition—Improving		24 per cent
Stationary		73
Getting worse		2
Work capacity—Incapable		23 = 19
Light work		29 = 24
Normal		67 = 56

IV PENETRATING WOUNDS

a WITH HERNIA CEREBRI *b* WITH RETENTION OF FOREIGN BODIES

Requests for information were sent to 206 cases of penetrating wounds of the head

I possessed, is in all other cases referred to in this paper, brief but moderately accurate notes as to the nature of the initial lesion, and the operative findings. This series of 206 cases includes 35 where a hernia cerebri developed soon after the injury, 42 in which foreign bodies remained in the brain substance too deep for attempts at removal, 19 with extensive extra- or subdural hæmorrhage (due to sinus injury middle meningeal hæmorrhage, cerebral laceration), and many others in which foreign bodies had been removed from the brain.

The serious nature of some of these cases may be demonstrated by a few examples —

Case 1 — Penetration of parietal region, shrapnel bullet retained in the very centre of the brain. Now suffering from slight headaches, nervousness, and some anæsthesia, but doing light work.

Case 2 — Penetrating wound of frontal region, followed by gas gangrene. Now well, except for occasional slight attacks of epilepsy.

Case 3 — Penetration of frontal region, followed by hernia cerebri and abscess of the brain. Foreign body remains deep in the base of the lobe. Well except for occasional slight seizures.

Case 4 — Penetration of parietal region, bullet removed from brain, followed by maggot infection of brain. Now working as a tram conductor.

Requests for information elicited the following replies —

<i>Headaches</i>	Yes, 196 = 95 per cent (severe 24, slight 172),	No, 10
<i>Insomnia</i>	Yes, 93	45
<i>Giddiness</i>	Yes, 152 = 73	,
<i>Nervousness</i>	Yes, 152 = 73	"
<i>Paralyses</i>	Yes, 73 = 35	"
<i>Fits</i>	Yes, 72 = 35	"
<i>Present condition</i> — Improving		61 = 29 per cent
Stationary		130 = 63 "
Worse		15 = 7 "
<i>Work capacity</i> — No work		79 = 38 "
Light work		49 = 24 "
Normal work		78 = 38 "

On analyzing further the 79 cases incapable of work in regard to the nature of their original injury, it was found that —

- 20 had suffered from hernia cerebri,
- 19 had foreign bodies in various parts of the brain,
- 3 had suffered from extensive abscess formation of the brain
- 5 had suffered from extensive intra- or extradural hæmorrhage,

and, on going further into the actual cause which, at the present time, incapacitates them from work, the evidence showed that —

- 25 suffer from paralysis and fits,
- 13 " " paralysis only,
- 17 " " fits only,
- 24 " " headaches, nervousness, want of concentration and loss of memory—18 being frontal injury cases

I might add that (1) In addition to the 35 cases of hernia cerebri, 22 other cases of hernia cerebri died in hospital—57 cases in all, with a mortality of 39 per cent, and (2) In addition to the 42 cases with foreign bodies retained who recovered, 19 others died in hospital, all from spreading infection of the brain and meninges—mortality 31 per cent.

(It should be noted, therefore that a death-rate from hernia cerebri of 39 per cent, and from retention of foreign bodies, with subsequent spreading infection of the brain, of 31 per cent, in base hospital at home, should be added to the death rate from these two conditions as occurring in hospitals in France etc.)

The table appearing at the end of *Section VI*, demonstrating the remote effects in relation to the severity of the lesion, shows clearly that penetrating wounds with hernia cerebri head the list—the most severe after effects and the least work capacity.

V PERFORATING WOUNDS

Inquiries from 19 cases brought the following replies —

<i>Headaches</i>	Yes, 17 = 89 per cent	No, 2
<i>Insomnia</i>	Yes, 10 = 53 "	
<i>Giddiness</i>	" 15 = 79 ,	
<i>Nerves</i>	" 12 = 63 "	
<i>Paralyses</i>	" 6 = 32 "	
<i>Fits</i>	" 3 = 16 ,	
<i>Present condition</i> —Improving		1 = 21 per cent
Stationary		15 = 79 "
Getting worse		0
<i>Work capacity</i> —Incapable		21 per cent
Light		26 1 = 79 per cent
Ordinary		53 1

These 19 perforating wounds of the head were of all varieties and directions, antero-posterior, lateral, and oblique, but in no case was there any retained metallic or osseous fragment, all foreign bodies had passed through or had been removed, nor were there any cases of hernia cerebri. In some it was to be concluded that there had been ventricular involvement.

It is rather astounding to note that the general after-result of this, the most extensive and serious injury of the head which is compatible with life, is followed by such, relatively satisfactory results. Headaches were less severe, and—with the exception of two cases—paralyses and fits less evident than in penetrating wounds.

	<i>Headaches</i>	<i>Paralysis</i>	<i>Fits</i>
Penetrating wounds	95 per cent	35 per cent	35 per cent
Perforating wounds	89 "	32 "	16 "

Furthermore, the work capacity of these perforating cases was of a high order.

	<i>Perforating Wounds</i>	<i>Penetrating Wounds</i>
No work	21 per cent	38 per cent
Light	26 1 = 79 per cent	24 "
Ordinary	53 1	38 "

It should be noted also that 21 per cent perforating cases stated that they were improving, 79 per cent remaining stationary, and that in no case were the conditions getting worse.

VI FRACTURED BASE

Fourteen cases of fracture of the base of the skull were admitted under my care. They presented no special features but were similar in all respects to those fractured-base cases that are seen in ordinary civil practice. As a rule they had been transferred to England soon after the injury, and on admission were suffering from headaches, mental sluggishness, ocular palsies, facial paralysis, monoplegia, aphasia, etc. Obviously, as in cases seen in ordinary hospital life, the basal fracture was of itself of little importance, the issue being dependent on the extent of associated intracranial injury. All these cases recovered, three or four after prolonged convalescence. No operations were performed on these cases other than occasional lumbar puncture for the attempted relief of the more severe grades of headache. In this late-history, the following results were obtained —

<i>Headaches</i>	12 = 85 per cent
<i>Insomnia</i>	9 = 64 "
<i>Giddiness</i>	13 = 93 ,
<i>Nervousness</i>	10 = 71 "
<i>Paralyses</i>	3 = 21 "
<i>Fits</i>	2 = 14 "
<i>Present condition</i> —Improving	3 = 21 "
Stationary	10 = 71 "
Getting worse	1 = 7 "
<i>Work capacity</i> —Incapable	2 = 14 "
Light	5 = 36 "
Ordinary	7 = 50 "

Table I —SHOWING THE REVOLUT EFFECTS OF GUNSHOT WOUNDS OF THE HEAD IN RELATION TO THE INJURY RECEIVED

NATURE OF INJURY'S	HEADACHES	INSOMNIA	GIDDINESS	NERVES	PARALYSIS	DETS	PRESENT STATUS			WORK CAPACITY		
							a	b	c	d	e	f
I —SCALP WOUNDS per cent	81	36	66	53	15	10	60	38	4	10	19	70
II —NON PENETRATING WOUNDS per cent	92	50	77	71	20	13	24	73	2	19	24	56
III —PENETRATING per cent	95	45	73	73	35	35	29	63	7	38	24	38
IV —PENETRATING, WITH HERNIA per cent	86	46	75	75	46	54	43	54	3	69	20	10
V —PENETRATING, WITH RETAINED FOREIGN BODIES per cent	93	31	76	71	33	33	21	67	12	43	36	22
VI —PERFORATING per cent	89	53	79	63	32	16	21	73	0	21	26	53
VII —FRACTURED BASE per cent	85	64	93	71	21	14	21	71	7	14	36	50
AVERAGE per cent	89	46	77	68	29	25	31	63	5	30	26	43

N B All figures are in percentages

VII GENERAL REMARKS ON THE REMOTE EFFECTS OF GUNSHOT WOUNDS OF THE HEAD, AND OTHER HEAD INJURIES WITH SPECIAL REFERENCE TO HEADACHES, FITS, RETAINED FOREIGN BODIES, PALSIES, ETC

"In addition to those fearful headaches from which I suffered, but which are now much better as the result of your operation, I get terribly nervous when going about the streets. I'm always thinking I'm going to see someone knocked down by a car. The noise of breaking of a car or cycle has a very bad effect on me. On such occasion my legs refuse to carry me, my knees go away from me. Concentration I find difficult, and in many cases impossible. Reading, except light stuff, is impossible. I've some terribly violent fits of temper, arising from trivial things, I find difficulty in controlling myself and lose control at most unexpected times. I sometimes have the feeling that I am going unconscious. Several people have thought at times that I was drunk. I'm T.T. I'm living a quiet life, and trying hard to fight above complaints."

This letter, one of the replies received to my inquiries, is typical of many others. It is so vivid a description of the general after-effects of a gunshot wound of the head that it is utilized as a text to this section.

Although the headaches are so frequently the outstanding, predominating feature in the case, it is quite clear that they are merely part of a more generalized state, one feature of a *syndrome*. I have dealt with this question in two previous publications,¹ and propose merely to recapitulate a few points.

In the syndrome are included the following conditions: (1) *Headaches*, (2) *Giddiness*, (3) *Insomnia*, (4) *Mental anxiety, depression, irritability of temper, and ready fits of violent passion*, (5) *General tremulous condition, shaking hands and uncertain gait*, (6) *Slight exaggeration of knee-jerks, with spurious ankle-clonus*, (7) *Fits ('fainting', epileptiform, and epileptic)*.

Since these symptoms occur with such frequency after gunshot wounds of the head, it is fair to argue that they all have some common cause, and I purpose trying to prove that, in many cases, at any rate they are associated with, and dependent on, a generalized condition of *cerebral oedema*. This statement is obviously not final and conclusive, but it can be proved, at any rate (a) that in most cases there is a great excess of cerebrospinal fluid and (b) that the removal and drainage of the excess brings about, almost instantaneously, a great relief of all symptoms.

It is necessary to take the most obvious symptom, *headache*, as a guide, noting carefully the presence or absence of excess fluid in those more severe cases that come to operation, observing the immediate results obtained, but withholding final judgement till such time shall have elapsed as will justify one in coming to more or less final conclusions. This is the course that I have adopted. The second of the two earlier publications on the subject was published in April, 1919, and three years have elapsed, or nearly so. The theories there advanced have received ample confirmation, not only from other cases of gunshot wound of the head, but also from many cases of civil practice, and from reports on cases operated on by other surgeons. Operation was only advised, and carried out, when my hand was forced in the worst type of case—in those patients who were 'fed-up', wearied with the incessant pain. In those earlier papers I was cautious in my prognosis, I was uncertain whether the great and immediate benefits would be maintained. Sufficient time has elapsed to enable more definite statement. The late results are here published of 40 cases of subtemporal decompression, where this operation was advised and conducted with the main object of headache relief. These cases are described and scheduled later. The results, taking them as a whole, are satisfactory.

1 HEADACHE

By reason of its frequency of occurrence its intensity and severity, its incapacitating effect on the patient, as also by its pathology and relief on decompression, headache forms perhaps the most interesting of the remote effects of head injuries, whether gunshot wounds or civil injuries. At the present time, four to eight years after the injury, 88 per cent of the cases from whom replies were received still suffer from headaches, varying

from slight and inconstant to severe and incessant, with, in these worst cases, periods of utter prostration, completely incapacitating them from work and rendering their lives miserable. The maximum percentage of headache was seen in penetrating wounds of the head, 95 per cent, the slightest degrees in cases of scalp-wound only, 81 per cent. Thus, by reason of the frequency and severity of headache, it is obvious that no apology is needed in considering the question fully.

In the great majority of cases the headaches date from the time of the original injury, and some patients still shudder at the horrible headaches they suffered from after regaining consciousness. Indeed, in many cases, the fact is noted in the history-card, and lumbar puncture was frequently carried out for its relief. The average after-history of these cases runs somewhat as follows: some weeks or months later, the headaches diminished in intensity and frequency, either finally reaching the slight and inconstant stage, so frequently observed at the present time, or showing little or no amelioration, remaining constant and severe.

In some, the earlier remission has been followed, during the last year or so, by a return to the earlier conditions, by relapse and exacerbation, often as the result of a return to work under the present difficult trade condition. Doubtless, family considerations, with attendant anxiety, have tended to add to the conditions aiding relapse. In others, after two or three years of comparative remission, the headaches have returned, sometimes in a severe form.

As seen at the present day, the patients suffering from the more severe grades of headache present a very typical facial appearance. It is easy to diagnose the condition as soon as the patient is seen—dour expression, fixed features, seldom relaxing and relapsing into smiles or more cheery expression, features outwardly expressive of headache—eyes of pain, with lids half-closed, frowning—the typical appearance of one suffering from headache. They are little interested in their surroundings, only too eager to accept any suggestion of operative treatment, with the hope of finding some relief from their condition. “I don’t care if you cut my head off” is a common reply to suggestion as to operation. The life is utterly miserable, and a conversation with the wife or sister is quite enough to clinch the argument as to whether operation is justifiable or not.

Many of these patients are soaked in bromides, etc., and their depressed state must be considered in that light. A period of remission from narcotics should be advised and carried out previous to final conclusions. I wish to urge, and point out, that operation should be considered only when all other measures fail. That has been my custom, otherwise I would have reported on 400 cases of subtemporal decompression instead of 40.

In the majority of cases the headaches are localized to the frontal region, sometimes to the occipital, more rarely to the vertex, with now and again a definite indication as to the localization of the pains to the region injured. Sometimes the pains are temporal or bi-temporal in position. Usually, however, “behind the eyes” is the complaint—“my eyes feel as if they were bursting.”

In regard to the time of onset, provided that the pains were inconstant, the headaches were most marked early in the morning, on waking. The next most frequent time of occurrence was about 5 o’clock. In all cases the headaches were intensified by exertion, bus rides, cinematographs, etc. Family rows were exceedingly conducive to further trouble. In the more severe attacks the patients retired to their rooms or their beds, only demanding quiet—curled up in bed, in the typical condition of cerebral irritation. Such cases were only too eager to be taken into hospital where they could be away from the noises of the house, rampaging children, etc.

In some cases the headaches are of a ‘cyclic’ character, recurring every two or three days, perhaps with a week’s remission, comparative freedom and then the attack, then freedom again till the next bout.

In relation to atmospheric and climatic surroundings these patients are regular ‘barometers’. A ward visit on a dull and heavy day with marked humidity, shows that almost every head-case has a headache, the more serious cases being miserable in bed.

On a bright and clear day, especially in the cool weather of spring and winter, the conditions are reversed, all are comparatively bright and smiling

Stuffy rooms, engineering workshops, with their noises and clangings, are bad for these patients. Open-air life, with light work, is markedly beneficial

The prostrating attacks are often accompanied by a slight rise of temperature, 99°–100°, with moderate degree of rise in blood-pressure, combined with some slowing of the pulse-rate. Marked distaste for food, with some nausea but no vomiting, and insomnia, accompany the attack

The discs rarely show any definite changes amounting in the more severe cases to some engorgement of the retinal veins. I have yet to meet a case with true papilloedema

In some cases the headaches are associated with an outburst of fits, usually of the epileptiform type, and it is remarkable that in some cases where there is a definite association of headaches and fits, the headaches very definitely lead up to the 'fit' development—the headaches gradually get worse till "my head seems as if it were bursting", then comes the fit. And so on, till the next occurrence

Now, as to the causation of these headaches. In the first paper published on this question, in 1918, it was stated that they were dependent on an increase of intracranial pressure, and that the increase of pressure was due, in the great majority of cases, to an excess of cerebrospinal fluid, the general sodden appearance of the brain and meninges giving rise to the term applied to the condition in general—*cerebral oedema*. This statement is proved by the two following facts: (a) The presence of excess fluid as found at operation, (b) The immediate relief produced by the operation—lessening of the intradural pressure in subtemporal decompression, and the provision of a door for the escape of excess fluid

a At the operation of subtemporal decompression, the routine operation carried out for the conditions present, the appearances of the brain are absolutely typical—the dura mater, when incised, allows of the immediate escape of excess cerebrospinal fluid, sometimes spurting out at high pressure in the form of a jet. When the dura mater is more extensively incised, the brain appears water-logged by excess fluid over the whole of the brain surface exposed, most marked in the line of the vessels as they run in their sulci. These vessels would appear, by reason of this fluid surrounding them, as white oedematous streaks. The fluid is seen to be exuding freely through the arachnoid, as drops or tears these running together in rivulets, these again coalescing, forming a pool at the lower angle of the wound, trickling away on the towels. On gentle pressure with gauze over the surface of brain exposed the excess fluid in the subarachnoid space is squeezed aside and, on the removal of the pressure, reaccumulates rapidly—altogether a very definite and typical picture, a very wet sponge. The removal of bone in a subtemporal decompression is so planned that it is easy to insinuate a broad spatula beneath the temporo-sphenoidal lobe, and when this is done the great accumulation of excess fluid at the base of the brain is well demonstrated—lifting up the brain, and then allowing it to fall back again each such manoeuvre being followed by the escape and evacuation of considerable excess cerebrospinal fluid

There can be no question, therefore, that in the majority of cases, there is a great excess of fluid

b When such excess is found at operation, the immediate results of decompression and drainage are exceedingly good. If the operation is conducted under local anaesthesia, and the patient is not too doped to be capable of recognizing conditions and surroundings, he will express himself, at the termination of the operation, as free from the old headache, and on one or two occasions the patients have sat up on the operation table and expressed themselves in terms of great gratitude. In any case, when seen the day after operation, the answer to inquiry is nearly always the same—the 'old headache has quite gone, even though it may have existed for one or more years. I am using the term 'old headache' advisedly, because the patient when questioned as to his condition will nearly always say that he has a headache—thus, on close questioning, is referred to the site of the wound. The decompression involves a fairly sensitive area and includes some section of the

temporal muscle—in consequence there is often some degree of local pain and discomfort. It is necessary, therefore, to discriminate between the 'old' and the 'new' headaches. The 'new' headache gradually lessens, and in the course of a week or ten days it also goes.

The effect on the patient generally is equally marked—previously dour and depressed, now bright and cheerful, hopeful for the future, dreading the possibility of return of those old and fearsome headaches, hopeful of having cast aside for ever the gloom that previously enveloped him, thankful for the relief given. All this shows that the evacuation of the excess fluid, by decompression and opening of the dural compartment, allows of the immediate relief of the headache.

Confirmation of these facts is gained by a study of the minority cases, operation being conducted in the anticipation that one is dealing with a case of cerebral oedema, but in which, at the operation, little or no oedema is found, and in place of this a slightly weeping, or dry, brain with bulging of the brain into the wound. These cases do not do so well—the headache relief is more problematical, both immediate and permanent. These minority cases obviously come under a different category, and are of a different pathological nature—I think they are examples of ventricular distention, due possibly to chronic meningeal thickening at the base of the brain, interfering with the outward passage of the cerebrospinal fluid.

There is, so far as my experience goes, no definite method of determining which condition is present, cerebral oedema or ventricular distention. The symptoms in the two cases are practically identical. It may be argued that preliminary lumbar puncture would settle the question, but it does not, for in many cases of cerebral oedema lumbar puncture has been negative to cerebrospinal fluid excess. There is no absolute necessity for intracranial excess fluid to be accompanied by spinal excess—it all depends on the conditions in the region of the foramen magnum—whether the communication between the intra and extracranial systems is free or not. Details as to lumbar puncture in relation to treatment will be considered later.

It is necessary now to consider the origin of this excess fluid. Here difficulties begin. I have argued that as the fluid is chemically, cytologically, and pathologically normal in every respect, that there are only two ways in which to explain the excess. Either it is *formed in excessive quantity and absorbed at an insufficient rate*, or it is *secreted at the normal rate and absorbed inefficiently*.

It is not difficult to determine which hypothesis is more probable. It is only necessary to consider the physiology of cerebrospinal fluid, its origin and course, to advance what is, at any rate, a very plausible theory. It is accepted (a) that the fluid is secreted from the choroid plexuses and lining ependyma of the lateral, third, and fourth ventricles, (b) that the greater quantity passes up over the surface of the cerebrum, in the subarachnoid space, and (c) that it is absorbed at the same rate as it is formed, into the superficial veins of the brain and into the lateral lacunæ of the superior longitudinal venous sinus. This passage of cerebrospinal fluid into the venous system may be the mere mechanical transmission of one fluid of a lower specific gravity and higher pressure into another through a permeable wall, or there may be some selective action on the part of the veins. In all probability the former simple explanation is the correct one.

Now there is a very significant factor present in most of the cases under discussion—a sufficient explanation for the accumulation of cerebrospinal fluid. In the majority of cases of gunshot wounds with brain destruction and subsequent fibrosis, in heat stroke by its effect on the surface veins in the brain, in cerebral malaria by the plugging and destruction of surface vessels, in the hæmorrhages of injury with brain contusion and laceration—there is in all a common final result: *the loss of a certain percentage of the surface brain area available for the absorption of cerebrospinal fluid*. This fluid, formed at a normal rate, is incapable of being absorbed in corresponding ratio, and in consequence there is an accumulation, shown in the minority cases by a condition of internal hydrocephalus (?), and in the majority cases by the condition of cerebral oedema, of which a description has been given. The fluid collects at the base of the brain, first in the

cisterna, and then accumulates in the meningeal spaces over the surface of the brain, where it is seen at operation

So far as I can see, the only weak link in my chain of argument lies in the fact that I cannot prove my case at the post-mortem table—the mortality after subtemporal decompression is nil, and I have been unable to prove my contention by microscopical and other evidence

Are there any other explanations available for this excess cerebrospinal fluid? Is it possible, for example, that they are similar to those described by Warrington² as intracranial effusions (serous) of inflammatory origin? Is it possible that the excess fluid is of inflammatory meningeal source? Malaria, heat-stroke, gunshot wounds, etc., might all conceivably lead to some form of chronic meningitis resulting in excess of fluid formation. But, in the cases under discussion, the fluid is always absolutely normal cerebrospinal fluid—there are no extracellular elements, and chemically the fluid is normal in all respects. I do not think, therefore, that these cases are in any respect of inflammatory origin—rather would I accept the view that some of Warrington's cases fall automatically into the group here described

The theory which I have advanced seems to be best adapted to the conditions as found at operation, and to the results obtained. The other symptoms of the syndrome are all to be explained on the same grounds—the mental depression and uncertainty, the general loss of muscle tone, the exaggeration of knee-jerks, etc., can all be accounted for by the sodden condition of the cerebral cortex, by loss of higher control

TREATMENT

The general lines along which treatment can be conducted in the *milder cases* are simple enough—and moderately efficacious

- 1 The patient should be advised to get a light job, preferably out-of-door work, such as poultry-farming, carrier, country delivery of letters, etc., under a considerate employer who will make all allowances for shortcomings, and for days off when the headaches are more incapacitating

- 2 Complete abstinence from alcohol

- 3 Regularity of bowels

- 4 Avoidance of exposure to the effects of heat

- 5 The provision of mild narcotics, which are to be taken during the periods of relapse. I prefer aspirin, pot. bromide, and chloral hydrate, of each 5 to 15 grains

- 6 The recumbent position in a quiet, darkened room, during the periods of prostration, if any

- 7 The avoidance of excitement of all kinds at all times—cinematographs, for example, are to be prohibited

- 8 The provision of a suitable pension, sufficing to allay the haunting fear of poverty

Under such conditions, these patients are usually enabled to carry on with reasonable comfort

The *moderately severe cases* are far more difficult to treat. In spite of rigorous action along the lines indicated above many cases are quite incapacitated from all work, and it is probable that operative measures, decompression, will be adopted more freely in this type of case—this statement being made in view of the generally satisfactory results of decompression

On the other hand all palliative measures should be tried first, and I believe that one of the most important is the provision of a living pension rate. This pension should be permanent, there should be no uncertainty about it, no periodic medical boards, etc.

In the *more severe cases* after careful consideration of all the circumstances, operative treatment (decompression) can be recommended with considerable confidence

Of the treatment of headaches by rectal sinuses³ I have but little to say. There is, however, about this method so much obvious impropriety, that further discussion is useless. In any case the benefit is purely of a temporary nature—it does not tackle the root of the disease

Treatment by lumbar puncture, though objected to on some similar grounds, requires more careful consideration. It is obviously a method of treatment that should be tried, and I have myself submitted it to a thorough test. I have used it so often that I am clear in my mind as to its general uselessness. I would go further, and say that it is also in many cases harmful. I would tabulate my reasons for these general statements as follows —

1 Cerebral œdema is not necessarily associated with any excess of fluid in the spinal meninges. Whether my views accounting for the excess cerebral fluid are correct or not, there can be no shadow of doubt that the continuity between the cerebral and spinal cerebrospinal spaces is commonly disturbed. Some of my most marked cases of plus cerebral fluid have been associated with minus spinal fluid—all depends on the meningeal conditions prevailing in the medullary region. In cases of marked cerebral œdema there may be such swelling and œdema in that region as to prevent the normal continuity between the two systems.

2 Even if the withdrawal of excess fluid by lumbar puncture brings relief, the effect is purely temporary—fluid collects again within a few hours, and the headaches are again as bad as ever.

3 Lumbar puncture frequently makes the headache much worse. The same effect has been observed in the treatment of cerebral and cerebellar tumours by lumbar puncture. The explanation is difficult though it is probably concerned in some way with the corking up of the medullary region. I have observed, in some few cases, that the immediate effect of lumbar puncture on a patient suffering from a severe attack of headache has been to throw him at once into a condition of agony.

4 Even if the lumbar puncture brings about temporary relief, the process cannot be continued *ad infinitum*.

In general I am quite opposed to lumbar puncture as a therapeutic measure in these cases. I carry out the measure once, submitting the fluid to chemical, cytological, and bacteriological investigation, and estimating its pressure by spinal manometer. I use Eve's cerebrospinal manometer (Fig 101).

‘The instrument consists of three hollow needles of sizes suitable for children and adults, large or small. These fit on to the stalk of a metal Y-piece.

‘To the two other branches of the Y-piece are fitted pieces of fine rubber tubing. One tube acts merely as an exit tube by which to draw off the fluid, and the other leads to the manometer. Both pieces of rubber tubing are provided with spring clips, so that the fluid can either be drawn off or diverted into the manometer. A detachable handle also fits on to the Y piece. This handle is so shaped that it can either be grasped, or held like a pen.

‘The three needles are tempered tough, so that the risk of their breaking *in situ* is avoided. It is important always to keep a wire in them except when in use.

‘The manometer consists simply of two lengths of glass tubing united by an inch of rubber tubing, and supported in a groove running the whole length of a folding metal scale. The groove is bridged over at intervals of an inch.

‘The hydrometer is capable of measuring the specific gravity of small quantities of fluid

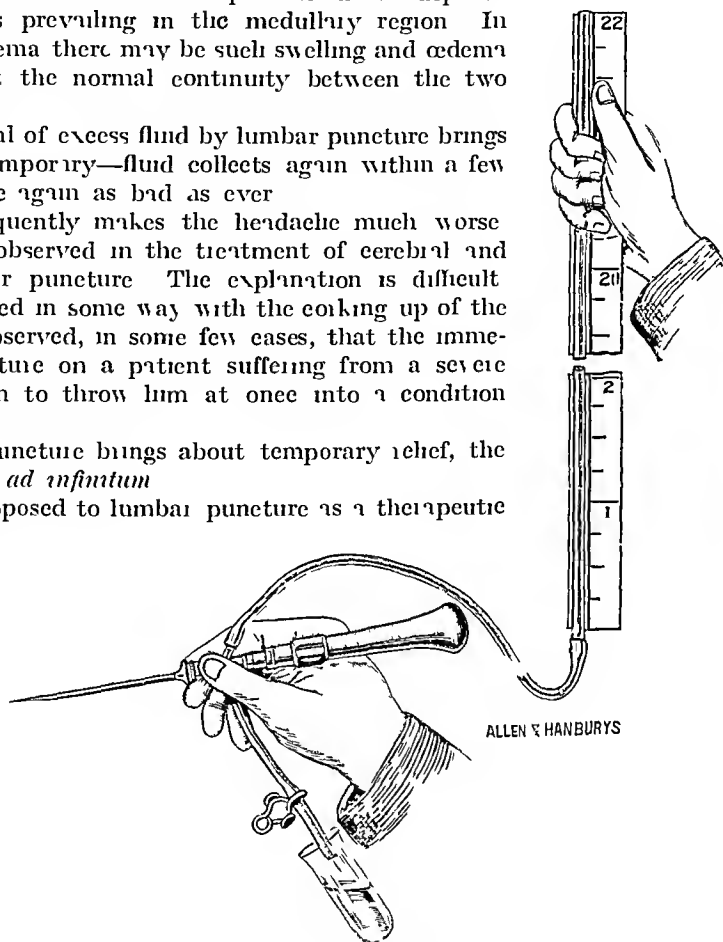


FIG 101—Eve's cerebrospinal manometer.

GUNSHOT WOUNDS OF THE HEAD

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(4 inches) The whole apparatus except the hydrometer is boiled and brought to the bedside in a shallow dish of boiled water. The apparatus is fitted together in accordance with the diagram, and any contained water is shaken out. "After introduction, the exit tube being already closed by a clamp, the fluid then rises in the manometer, and the pulsations due to the pulse and respirations are seen. If the fluid fails to appear, it may often be coaxed by 'milking down' the rubber tube. "The pressure is recorded when the zero of the manometer is held at the same level as the needle. If the manometer is held vertical, it shows the pressure of the cerebrospinal fluid in terms of a column of water so many inches high."

2 GIDDINESS AND NERVOUSNESS

Giddiness, present in 77 per cent of cases, and nervousness, in 68 per cent, though slightly improved under bromide treatment, are both but little benefited by decompression operation. It is necessary, therefore when carrying out decompression operations for the relief of headache, to make it quite clear to the patient that the two minor ailments, giddiness and nervousness, will be but little improved, if at all. They are both, however, of far less disabling nature than headaches. Plating of the defect of the skull may, in my experience, lessen the general nervous symptoms, but it has little effect on the giddiness.

3 PARALYSIS

It was to be expected that a considerable percentage of the head cases would suffer from permanent paralysis of varying degree. The percentage is less than expected, but quite sufficiently serious (29 per cent). The following table shows the percentage rate according to the nature of the injury incurred—

	Per cent
Penetrating wounds with hernia cerebri	16
Penetrating wounds	35
Penetrating wounds with foreign bodies retained	33
Perforating wounds	32
Fractured base	21
Non-penetrating wounds	20
Scalp wounds	15

Penetrating Wounds with Hernia Cerebri—In most cases the injury was received over the anterior and lateral aspects of the skull. Patients with occipital and cerebellar hernia seldom survived, especially fatal. When the protrusion involved the motor cortex, hemiplegia resulted, and in many cases the paralysis is of a permanent nature, but it may be added that in some cases the ultimate result was extraordinarily good—the patient recovering almost full power.

Penetrating Wounds, with or without Foreign Body Retained, come next in the list of cases presenting permanent paralysis, but it is interesting to note that the retention of a foreign body does not appear in itself to present any additional paralytic disability. 33 per cent with such bodies retained, 35 per cent where none are present, whether removed early or at a later date. In other words, the paralysis results from damage done by the penetration of the body itself. This point must not be laboured—but it should certainly argue against the early judicious removal of foreign bodies is advanced—but it should certainly argue against the retention of the body itself. This point must not be laboured—but it should certainly argue against the retention of the body itself. This point must not be laboured—but it should certainly argue against the retention of the body itself.

Perforating Wounds, 32 per cent, come next in the list, the lessened paralytic rate, being due in part to the high death rate of this class of injury—only the slighter cases recover. **In Fractured Base, Non-penetrating Wounds, and Scalp-Wounds**, the paralytic rates are 21, 20 and 15 per cent respectively. They are obviously dependent on brain lacerations and

* I think that under normal conditions the fluid rises to a height of 6 to 8 in. When under excessive pressure I have seen the fluid running from the top of the manometer 16 to a height of 24 in.

contusions, surface hemorrhages, both external to and within the dura mater. It is a debatable point, but one certainly worth consideration, as to whether some of these cases would not have benefited by early operation, with the hope of evacuating extradural or intradural blood-clot (see SCALP-WOUNDS, p. 94).

A large number of these paralytic cases have improved under treatment, massage, radiant heat, re-education, etc., but many remain more or less totally incapacitated. The proportionate improvement in upper limb, lower limb, and face has followed the normal course as regards the degree and rate of recovery in the three regions mentioned—face recovering first and most, followed by the lower extremity, the upper always lagging behind. Recovery has been exceptionally poor after injuries of the superior longitudinal sinus—Sargent's longitudinal sinus syndrome.⁴

Allusion will be made later to operative treatment, but it would be wise to state here that plating or closure of the defect in the skull brings about, in my experience, but very little, if any, benefit for this type of case.

4 FITS

The prevalence of fits after war injuries of the head is of the utmost importance. Early in the war it was stated that the percentage of cases in which fits developed was very low. That also was my experience, but I was sceptical as to whether the picture would not change. In this series, fits were reported in 25 per cent of cases, in the following percentages according to the lesion—

	Per cent
Penetrating wound with haemorrhage	54
Penetrating wound	33
Penetrating wound with foreign bodies retained	33
Perforating wound	16
Fractured base	14
Non penetrating wound	13
Scalp wounds	10

This list should be compared with that illustrating the rate of paralysis in relation to the site and nature of the injury—there is a close resemblance.

Nature and Frequency of Fits—On analysis, it was found that the fits could be divided into four groups, viz.—

	Per cent
Epileptiform	57
Jacksonian	23
Fainting	16
Slight and uncertain	4.5

It is noteworthy that the more severe types of fit were associated with the more serious lesions. Thus, in non-penetrating wounds there was 1 case of Jacksonian epilepsy, 6 cases of epileptiform seizures, 4 of fainting, and 5 of a slight nature, whilst in penetrating wounds there were 20 cases of Jacksonian and 42 of epileptiform fits, 2 only of the fainting type, and 7 of the slight variety.

The term 'epileptiform' is used for those generalized fits in which there was sudden loss of consciousness, followed by struggling, often of a violent description, and sometimes necessitating restraint with the help of three or four assistants, the patient passing urine involuntarily, biting the tongue and remaining unconscious for varying periods of time, a few minutes to hours.

The term 'fainting' is used for a type of fit of which I have had little previous experience—a sudden relapse into the dream-state, with no biting of the tongue etc., enduring a few minutes only, and leaving the patient tired, uncertain as to what has happened, and complaining of severe headache—attacks of petit mal of sorts.

Perhaps these 'fainting' fits, and other types of fit, may be explained by a "vaso-constriction reaction of the minute vessels in the cortex, thus causing anæmia of the brain and, in consequence, a greatly increased sensitiveness to internal and external stimuli, the so-called 'Stokes-Adams syndrome'."

In the treatment of these fit cases, my experience is such that I do not consider operative measures are of much avail, though in some instances the plating of the defect (after Sargent's method) has brought about some benefit. Prolonged and assiduous treatment with bromides, luminal, etc., should be carried out, combined with admission to hospital during the more severe stages. It is remarkable how the condition can be controlled when the patient is properly looked after. This improvement is due, not to the medical treatment of a neurasthenic case, but to the transference of a patient inclined to fits from the economic and family difficulties and exciting incidents of home life to the quiet of hospital, with its systematic and sympathetic treatment, associated with that confidence in the medical man in charge of the case which is so essential.

Luminal, first recommended to me by Sir Frederick Nott, has received a good trial. On the whole, I am inclined to believe that it is the most efficacious drug in fit control, given as a rule in $2\frac{1}{2}$ -gr doses night and morning. I note, in a recent paper,⁶ that stress is laid on the establishment of tolerance in patients under luminal, the frequent necessity of dosage increase to obtain control, and the bad effects produced by sudden withdrawal of the drug. I have not noticed these effects myself, though I have recently seen one of my patients under luminal who took five times the dose by accident and who was brought to the hospital in a state of violent excitement closely simulating over-indulgence in alcohol. In any case the drug must be given with caution, its effect carefully noted, and the patients warned against overdose.

GENERAL REMARKS ON WORK CAPACITY

I should like to preface this section by a statement to the effect that, of the 400 to 500 cases, there was not a single case in which the patient stated that he could work and could not get work. Whether this applies to the immediate present, I cannot say. My investigations have shown clearly that the slackers are but few in number. Here is a table showing how the men are working, in relation to their wounds and general disabilities.

	No work Per cent	Light work Per cent	Heavy work Per cent
Scalp wounds	10	19	70
Non penetrating wounds	19	24	56
Penetrating wounds	38	24	38
Penetrating wounds with hernia cerebri	69	20	10
Penetrating wounds with foreign bodies retained	43	36	22
Perforating wounds	21	26	53
Fractured base	14	36	50

Is it not rather extraordinary that, in the case of penetrating wounds with retained foreign bodies in the brain substance, 58 per cent should work, light or heavy, and that in perforating wounds, 79 per cent should be earning their living in whole or in part? As another example of the work capacity of these patients, is it not marvellous that one of my patients, with a shrapnel bullet in the very centre of the brain, should be working eight hours a day at pattern-making?

RETAINED FOREIGN BODIES

It will be noted that some arguments have been deduced from time to time against the removal of foreign bodies from the brain substance. This statement must be accepted advisedly. All experience shows that the immediate removal of foreign bodies should be encouraged provided that in the process of removal, every precaution be taken against increasing the damage already incurred by the penetration into the brain of the foreign body. It is clear however that *such bodies may remain encysted in the brain substance without producing any harm whatever*. On the other hand, there is some remote chance of a flare (see POSTSCRIPTUM p. 125). These flares, however, are very rare, and it is wise to advise caution in the ranks of the younger generation, as regards the removal of the foreign body both early and late.

VIII DECOMPRESSION, WITH DETAILS OF 40 CASES

Subtemporal decompression is the routine operation carried out for the relief of headache. This operation has but little, if any, effect upon the other symptoms of the syndrome—giddiness, insomnia, nervousness, etc.—and all my cases of decompression 40 of which are appended, were carried out with the main object of relieving headache.

As regards the rationale of this decompression operation, I believe that in the great majority of cases the headaches are due to increased hypertension the result of excess cerebrospinal fluid and that the rational treatment is to trephine over some 'silent' area of the brain, preferably on a level with the base of the brain (for more efficient drainage), in some situation where the scar is inconspicuous and where the osseous defect can be protected with muscle-flap. The excess fluid would be permitted a means of escape into scalp tissues where it is more readily absorbed, the

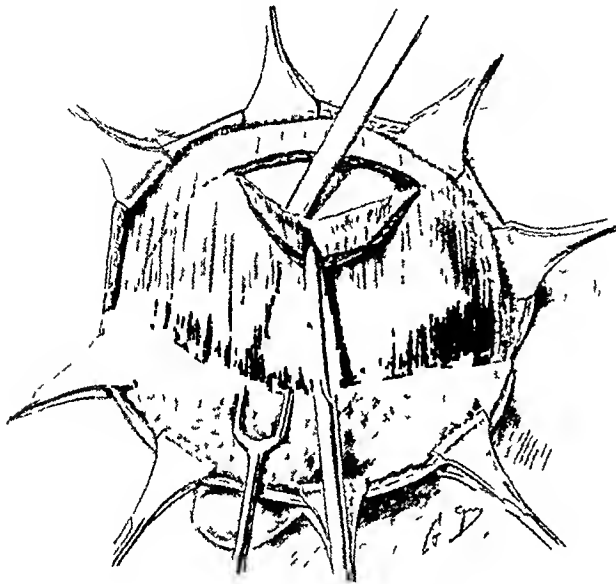


FIG. 102—Subtemporal decompression. Stage 1. Hemostatic forceps applied to the scalp margin. Temporal muscle incised and stripped from the bone by periosteal elevator.

intradural hypertension should be relieved at once, and immediate benefit obtained so far as headache is concerned.

We knew something about 'oedema of the brain' long before the war, but the first of the series of cases on which this paper is based was operated on in India—a soldier from Mesopotamia, invalided to India with ferocious headaches after heat-stroke.

I think I adopted Cushing's method of decompression, the inter-musculotemporal route, and this course I have adopted a few times in subsequent cases usually those of the milder description. It presents some advantages over the method described below, but it does not permit of the degree of exposure required for the necessary brain examination; the field of operation is cramped, and there is always some risk of damage to the anterior or main branch of the middle meningeal artery, more especially where that vessel runs in a canal or groove in the bone. Consequently, I am accustomed to decompress the temporal region after the following manner—

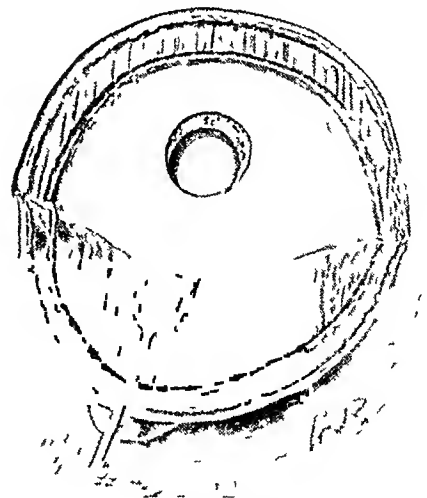


FIG. 103—Subtemporal decompression. Stage 2. Temporal muscle turned down. Bone trephined over centre of exposed area.

The apex of the ear is stitched to the cheek, to get it out of the way, after which a curved incision is made, the convexity of which lies about one inch below the temporal crest, the ends curving downwards as seen in the illustration (Fig 102)

The incision should be commenced at the summit of the curve, and carried down to expose the temporal fascia—about one inch at a time, hæmostatic forceps being applied to either cut edge of scalp, thus obtaining a practically bloodless field. When the incision is completed, the forceps are removed one by one, and bleeding points secured and tied in the ordinary manner.

The skin and subcutaneous flap is turned down for about three-quarters of an inch, when the temporal fascia and muscle are divided to the bone, again at the summit of the wound, about half an inch below the margin of the scalp incision, the temporal muscle is then seized with Lane's forceps, and the muscle stripped up from the bone with a periosteal elevator, down to the level of the

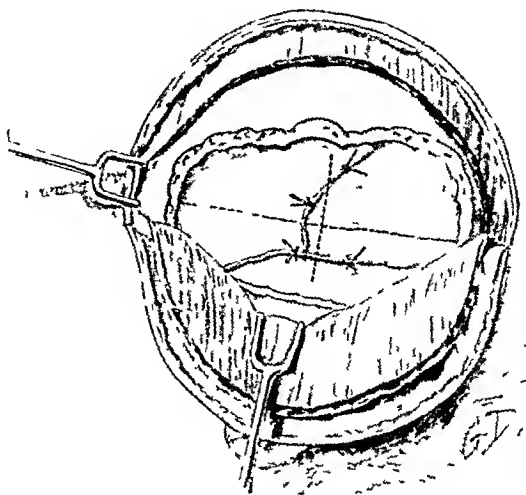


FIG 101—Subtemporal decompression Stage 3. Bone cut away in the forward, backward and downward directions. Location of posterior branch of middle meningeal artery and line of dura incision.
N.B.—In practice more bone should be cut away than is represented in the illustration—more especially in front or below, in relation to the retractors.

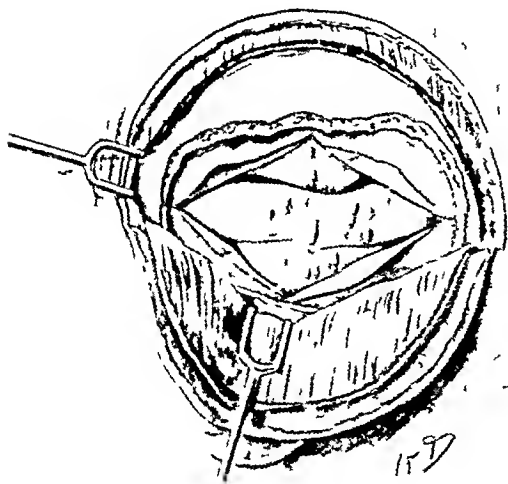


FIG 102—Subtemporal decompression Stage 4. Diagrammatic representation of the edematous and swollen brain, showing the area exposed and the drainage space provided by the operation.

but more especially in the downward towards the base of the skull and base of the brain,

being divided in front and behind with the scissors, in a line with the skin incision. Any bleeding points are secured. The pericranium is stripped away with the muscle. A half-inch trephine is applied to the centre of the bone exposed, and the disc removed (Fig 103). There is no fear of damaging the middle meningeal artery, the trephine area being situated in the angle between its anterior and posterior branches. The appearance of the dura exposed usually shows whether the diagnosis is correct or not and whether a condition of cerebral œdema is existent or not. In this condition, the dura itself is seen to be œdematous, and to have lost its sheen and translucency. As a rule, moreover, it is not tight nor does it bulge, but pulsation is absent.

Before opening the dura mater, the bone is nibbled away in the forward and backward directions,

towards the level of the zygoma and attachment of the ear. The muscle flap is well held up, and the attached muscle in front and behind well retracted so as to allow of the free application of the nibbling forceps, an aperture being framed which is not less than 2 inches in the anteroposterior direction, and 1½ inches in the vertical, the aperture lying throughout underneath the temporal muscle, and reaching down to the base of the skull. When nibbling in the anterior direction, care must be taken to avoid injury to the anterior branch of the middle meningeal artery—the posterior will cross the area of dura exposed, in the horizontal direction.

The lower the aperture is situated, and the nearer to the base of the brain, the more free will be the escape of excess cerebrospinal fluid when the dura is opened.

The posterior branch of the meningeal artery will require ligation in two places before the dura is opened. This is done with an intestinal needle, threaded with fine silk, the needle being passed so as to surround the vessel without injuring the underlying pia-matroid. If the point of the needle enters the subdural space, when a condition of oedema is present, fluid will escape through the needle-hole, sometimes in a fine spurt, thus establishing the diagnosis even before the dura is properly opened. The dura is now incised, in a crucial manner, and slit up in the four directions, right up to the margins of the bone aperture (*Figs 104 and 105*).

If the conditions are as expected, cerebrospinal fluid escapes freely, and a blunt spatula, insinuated beneath the temporosphenoidal lobe, lifting it up, will allow of the escape of more fluid.

The dura is left open. The temporal muscle is approximated with a few catgut sutures, and the fascia sewn up so far as circumstances permit. The skin is sutured with fine salmon-gut, and the wound closed without drainage. The excess fluid escapes into the tissues of the side of the head and face—sometimes leading, in marked cases of oedema, to considerable oedema of the face. This condition soon mends.

The wound is painted over with picric or iodine, and layers of gauze are laid firmly and evenly over the wound.

The stitch is removed from the ear, the ear smeared with ointment, and the dressings are secured with bandages, care being taken to see that the ears are flat, not bent over.

The patient is put back to bed, in the sitting-up position, and kept lightly under the influence of morphia for the first twenty-four hours. The wound is redressed completely the day after the operation.

General or Local Anæsthetic?

There are points in favour of either method. The main point in favour of local anæsthesia is related to the question of vomiting—the increase of intracranial pressure associated with the act of vomiting, and the fact that the brain is now unsupported in the region of the aperture, renders it highly desirable that the vomiting element should be eliminated, if possible. There can be no question that vomiting is of less likely occurrence after a local anæsthetic.

On the other hand, there can be no question that the operation can be conducted more freely and more easily under a general anæsthetic, and if complications should arise, for example troublesome bleeding from a meningeal vessel, the difficulties can be overcome more readily.

Local—If the operation is to be conducted at 1.30, the patient at 1 o'clock receives a hypodermic of

Morphine	gr 1-4
Atropine	gr 1-100
Hyoscine	gr 1-100

and a second hypodermic, same strength, at 1.15.

These injections should be given when the patient is quiet in the anæsthetic room, on the operating table. The eyes should be covered and the ears filled with wool. Then at 1.30 he is wheeled into the theatre and the 'local' anæsthetic given. I am accustomed to use Gray's syringe, with a 2 per cent solution of novocain, freshly prepared, to each

10 cc of which are added 5 drops of a 1-500 solution of adrenalin. The solution is injected subcutaneously in the line of the proposed incision, and along the base of the flap, blocking the operation field (Fig 106)

I must admit that I was much surprised, when doing my first case under this method, to find that not only was the cutaneo-muscular flap formation painless, but that trephining and enlargement of the aperture were painless also. During the first few minutes of the operation the patient is often nervous, and during the trephining may express some resentment, but I have never heard the patient complain of actual pain. The dural incision is passed unnoticed, as also is digital examination of the brain.

Of course the psychology of the patient must be taken into consideration, and it is desirable to have a skilled anaesthetist present in case his services should be required.



FIG 106—Infiltration of the operation area with novocain and adrenalin solution

As to the *side on which the operation should be carried out* the right side, in right-handed individuals, is the side of choice, but I never hesitate to operate on the left side—the possibility of interfering with Broca's motor speech area should not arise. I am sure, however, that one should be influenced by any localization of headache—operating on the side on which it is the more severe. Also, it is advisable to operate on the same side as that on which the injury was received.

After-Treatment—It is most desirable that these patients should be kept in bed for not less than three weeks after the operation. This is often very difficult to carry out in practice for being relieved of headache after months or years of suffering, patients become very intractable. It is desirable also, that there should follow a long period of convalescence at some quiet spot, for three months or more.

Remote Results of Subtemporal Decompression—The immediate benefit, amounting usually to complete relief from the old headache, is dependent on the escape of the pent-up fluid from the intradural space. It would be anticipated, however, that as the wound heals and scarring occurs the aperture would become closed with scar-tissue, with a return therefore of the old headaches. I expected this result—it ought so to be, and to some extent it is so—but not to the degree anticipated. On following up the various cases two to four years after operation I have been gratified at the ultimate results in general. There have been some more or less complete failures, and in quite a number the headaches returned again after a few months though it is most exceptional for them

at all to resemble the fearful and constant type experienced previously. In some cases the cure is apparently permanent. A complete account with after-results is appended, and it will be noted that the unsatisfactory cases presented very serious primary lesions. In weighing the pros and cons of the operation it should be noted also that the 40 cases reported were the most severe cases of war headache that I have encountered in the last six years.

Choice of Case—The following points should be considered before advising operative procedures —

1 It matters not how long the headaches may have persisted, in fact, the longer the more likely the success.

2 Constant headaches, more especially when accompanied by frequent periods of exacerbation—being associated as a rule with marked oedema—are more amenable to treatment than the inconstant cases.

3 The prognosis is better —

- i When the headaches are accompanied by that facial appearance of depression and misery to which allusion has already been made
- ii When the patient is 'fed-up' with his trouble
- iii When marked excess of cerebrospinal fluid is found on lumbar puncture
- iv When there is no bleeding at operation and no tendency to hæmorrhage
- v When marked cerebral oedema is found at operation
- vi When the operation is followed by little or no vomiting
- vii When the patient is able and willing to follow out the after-treatment prescribed

An analysis of 40 cases of subtemporal decompression shows that the operation was carried out for the following conditions —

Headache following on gunshot wounds of the head	24 cases
" " " concussion, etc	6 "
" " " malaria and heat stroke	3 "
" " " previous injuries, aggravated	3 "
" " " previous fits, aggravated	4 "
	<hr/>
	40

It is interesting to note that headaches in general, apart from these 40 decompression cases, were associated usually with non-penetrating and grazing wounds of the head, cases in which the primary operation, if any, was of but slight decompressive nature. There was a considerable revulsion of feeling against Sargent's extensive craniectomies in the early stages of the war, but I am quite certain that headaches are of more frequent occurrence in those cases where the surgeon has abstained from operation or carried out a very minor form of bone removal.

This would suggest that, although primary excision of the scalp-wound and primary suture are advisable in general, it is best to carry out also a fairly extensive removal of bone. (*See below*)

SYNOPSIS AND CRITICISM OF 40 CASES OF DECOMPRESSION (*details in Table*)

1 Cause of Symptoms —

Secondary to gunshot wound of the head	24 cases
" " concussion	6 "
Aggravation of pre-existent headaches, due to injury or disease	7 "
Secondary to heat stroke, cerebral malaria, etc	3 "
	<hr/>
	40

GUNSHOT WOUNDS OF THE HEAD

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2 Operation carried out for the relief of —
Headache only
Headache and fits

25 cases
15 "
—
40

3 Operation —
Left subtemporal decompression
Right subtemporal decompression
Bilateral subtemporal decompression

32 cases
6 "
2 "
—
40

4 Conditions found at operation —
Marked oedema
Moderate degree of oedema
Slight oedema
Practically no oedema, mainly general tension of the brain,
characterized by bulging

21 cases
5 "
2 "
—
12 "

5 Immediate effect of the operation on the headache —
Very great immediate relief, amounting in most cases to complete
relief
Improvement, fairly satisfactory

12 "
—
40

6 Remote effect of the operation on the headache —
Completely free
Slight headaches
Moderate and severe headaches

30 cases
10 "
—
40

(Note that operation was only advised and carried out for the very severe cases)
7 Remote effect of the operation on the fits —
Patients who have had no more fits or else fits of quite slight nature
Patients who have fits much the same as before, though in some
the fits may be less violent and frequent

4 cases
27 "
9 "
—
40

(I have reason to believe that this relatively satisfactory result has not been borne
out by subsequent cases)
8 Work capacity of the patient upon whom subtemporal decompression has been carried
out —
Doing ordinary work
Light work
No work
Uncertain

6 cases
9 "
—
15

17) 27 = 67.5 per cent
10)
11) 13 = 32.5 per cent
2)
—
40

This working capacity should be compared with the general average, viz —
Scalp wounds
Non penetrating wounds
Penetrating wounds
Penetrating wounds with hernia
Penetrating wounds with retained foreign bodies
Perforating wounds
Fractured base
(Note that the 40 cases submitted to decompression operations were previously
totally incapacitated)
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Per cent
89
80
62
30
58
79
86

Table II—LIST OF 40 DECOMPRESSION CASES

DATE AND NATURE OF INJURY	SYMPTOMS	OPERATION AND DATE	PRIOR STATE AS TO HEADACHES	DID OPER. BRING RELIEF
Epilepsy and headaches since 7 years old Aggravated by service in Mesopotamia	Severe epileptic fits with constant and severe headaches	Left subtemporal decompression, Sept., 1916 Œdema slight	Slight occasional	Yes
Epilepsy and headaches since childhood Aggravated by service in Gallipoli and Mesopotamia	Severe epileptic fits with prostrating headaches	Left subtemporal decompression, Sept. 1916 Marked œdema	Slight occasional	Yes greatly satisfied
Depressed position of vault in 1911 Headaches previously slight became much aggravated by service in Mesopotamia	Persistent headaches with periodic prostration Insomnia Occasional vomiting	Right subtemporal decompression, Nov., 1916 Marked œdema	Occasional severe, generally slight and inconstant	At first very poor now good
Previous history of fits and headache, much aggravated by service in Mesopotamia	Severe and prolonged fits Persistent and severe headaches Periodic prostration	Right subtemporal decompression Jan., 1917 Marked cerebral œdema	No headaches	Yes
Meningitis(?) when 14 Life miserable Worse after service in Mesopotamia	Chronic and persistent headaches with exacerbations	Left subtemporal decompression, Jan., 1917 Marked œdema	None	Quite well
(?) Heat stroke, (?) Cerebral malaria in Mesopotamia	Violent epileptiform fits, severe headaches, often with prostration	Left subtemporal decompression Feb. 1917 Some œdema marked general bulging	None	Yes
G S W Mastoid Dec., 1916 depressed, non penetrating	Persistent headaches	Left subtemporal decompression April, 1917 Œdema well marked	Severe at times	Somewhat better than before operation
Heat stroke 1915, in Mesopotamia	Violent epileptiform fits with severe and persistent headache	Right subtemporal decompression August 1917 Marked tension, but little œdema	At times	Yes good first not so
G S W Left frontal, penetrating June, 1917	Very severe and constant headache Severe exacerbations	Left subtemporal, Dec., 1917 Marked œdema Right subtemporal Oct., 1918 Less œdema	Varying from very slight to moderate, inconstant	Considerable relief first operation after second
G S W Frontal, non penetrating Oct., 1917	Persistent headaches, with exacerbations	Left subtemporal March, 1918 Œdema moderate	Slight occasional	Yes
G S W Left temporo parietal removal of bullet from brain Feb., 1917	Headaches aphasia hemianopsia Fits	Removal of bullet March, 1917 Left subtemporal March 1918 General bulging	Slight occasional	Yes entire headache-guidance
G S W Left temporal and occipital regions, severe brain laceration and numerous small foreign bodies remaining in brain	Violent headaches, with screaming fits Streptococcal meningitis	Right subtemporal April 1918 General bulging	Great relief at first then recurrence, and now better again	Yes
G S W Left temporal July 1916 with laceration of brain	Constant headaches, with occasional epileptiform fits Dull mentally	Left subtemporal May, 1918 General œdema	Slight occasional	Instant relief
G S W Fronto parietal penetrating 12/4/18	Very severe and constant headaches with exacerbations	Left subtemporal decompression 31/5/18 Moderate degree of œdema	Occasional	Yes

GUNSHOT WOUNDS OF THE HEAD

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TH PARTICULARS AND END-RESULTS	IF AND WHERE ARE THE HEADACHES	ANY OTHER COMPLAINTS	ANY FITS?	WORKING?	REMARKS FROM PATIENTS	CONCLUSIONS
	Mostly at night	None	None	Labourer		Good result
	ornings frontal	None	None	Grocer	"I feel practically as well as as when I left England"	Good result
	s on getting specially in dull ny weather	None	None	Railway carriage cleaner	"I was sent to France in 1918 which did me no good" Now much better	Good result
		Giddiness	Occasional slight	Provision trade	At times all right, but after excitement get my headaches again	Fair result
		None	None	Commercial traveller	"Instant relief and all right so long as I wear a protector over the hole"	Good
	in temple especially work and worry	Depressed	None	Farm hand	"Quite well"	Good
	in the mornings late in the even	None	None	Light engineering	"Not so strong as I used to be, and memory bad"	Moderate
	and after ex ment and worries	Sleepless at times and nervous	None	None	"Very susceptible to changes of weather especially moist heat"	Moderately good
	in the left	None	None	Light farming but very little	"Better and improving"	Fairly good
	across the	None	Two fits only	Army Pay Office	In good health head aches not worth worrying about and sleep well	Good
	and night but in region of	None	Two fits	Electric railway	Not satisfied	A very severe case results satisfactory on the whole
	trim and bus vs and in lot	None	None	None		Result good in consideration of nature of injury
	aporal region	None	None	Trying to get work		Good
	Motor driving				I don't think I should be writing except for your work The horrors of those old headaches come times come back to me	Good

Continued on next page

Table II—LIST OF 40 DECOMPRESSION CASES

DATE AND NATURE OF INJURY	SYMPTOMS	OPERATION AND DATE	PRESENT STATUS TO HEADACHES	DID OPER. BRING REL.
Concussion in 1905, headaches frequent and aggravated by service in India 1916-1917	Constant headaches with frequent and violent exacerbations 'No interest in life' Some fits	Left subtemporal decompression 15/8/18 Marked cerebral oedema	More or less constant	Not q
G S W Right occipital, penetrating, 18/4/18	Constant headaches and apathy	Left subtemporal decompression 30/8/18 Very marked oedema	Slight occasional	Yes
G S W Right frontal penetrating Date ?	Constant headaches, with exacerbations Depression and fits	Left subtemporal decompression 10/11/18 Marked oedema	Slight occasional	Yes
G S W Occipital (Palestine) 29/11/17 'injury to brain	Severe and persistent headaches bi temporal, culminating at vertex	Left subtemporal decompression 5/10/18 Moderate degree of oedema	Slight occasional	Yes
G S W Right frontal 3/10/18 No operation	Apathetic and listless Continuous headache, usually very severe	Left subtemporal decompression 19/10/18 No oedema, bulging only	Slight occasional	Yes
G S W Right parieto occipital with extradural hemorrhage and with foreign body remaining in right occipital lobe 28/7/18	Constant headache, with periods of vomiting 'Woke up with it, and go to bed with it' Jacksonian fits	Left subtemporal decompression 1/11/18 Marked oedema	Slight constant	'I feel better for the operation' did me a lot of good
G S W Right parietal penetrating in Mesopotamia 8/11/17	Constant headache with frequent prostration	Left subtemporal decompression 7/11/18 Marked oedema	Slight occasional	Yes
Concussion (blown up), 14/8/18	Constant headache with periods of prostration	Left subtemporal decompression, 14/11/18 Marked oedema	Slight occasional	Yes
G S W Left mastoid penetrating and fracture of base of skull	Constant headaches	Right subtemporal decompression 22/11/18 No oedema marked bulging	Slight occasional	Yes
G S W Right parietal non penetrating 9/9/18	Constant headaches with periods of severe exacerbation	Right subtemporal decompression 7/12/18 No oedema some bulging	Slight occasional	Yes operation brought great relief
G S W Right orbito frontal penetrating Date ?	Constant headaches dull mentality with frequent exacerbations	Left subtemporal decompression 19/12/18 Some oedema some bulging	Occasional	'Not so bad as before operation'
G S W Frontal, penetrating Dardanelles 12/8/16	Headaches commenced in 1917 gradually becoming more severe Weakness of left leg	Left subtemporal decompression, 27/12/18 with marked oedema	Slight occasional	Yes
G S W Left fronto parietal penetrating 17/7/18	Constant dull headache never free	Left subtemporal decompression 1/2/19 Marked oedema	Very slight constant	Yes
Blown up depressed fracture left frontal dura intact, 24/11/17	Constant headaches with exacerbations	Left subtemporal decompression 12/9/18 Some oedema but marked bulging	Constant slight severe occasionally	'Some relief but rather much anticipated'
Head injury in Mexico 1916 Headaches and fits since aggravated by service in France	More or less constant headaches Mild epileptiform seizures	Left subtemporal decompression 15/4/19 Marked oedema	Severe occasional	'Operation successful' far a headachy concern

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WITH PARTICULARS AND END-RESULTS—continued

IF AND WHERE ARE THE WOUNDS?	ANY OTHER COMPLAINTS	ANY FITS?	WORKING?	REMARKS FROM PATIENTS	CONCLUSIONS
times one side at times the opposite	Depression Malariad attacks	Jacksonian epilepsy right	Light and occasional at wool and cotton mill	—	Rather moderate
no specified	None	None	—	—	Good
time sides and top head	None	One fit	Returned to Australia	—	Good
bending or after motion across eyes	None	None	—	—	Moderate
severe weather or cold in left nape	Do not feel as well as soon after my operation	Two fits	Railway	'Never felt better'	Good
times of weather	Malaria	None	—	'Immediate results were good, but I have gone back'	Moderate only
my work brings on headaches at other times well	None	None	None	—	Fairly good
sultry weather and excitement	Facial paralysis left	None	Locomotive	Improving "	Fairly good
my weather rushing out in temple	None	None	Coil winding	—	Fair only
not on bending	Insomnia	None	Motor mechanics	Since operation my head has not given me half the trouble which existed before I seem to be brighter and better	Fairly good
reaction of wound special exciting mind except willing	None	None	Part time pit bank	In the pink one minute and down in the dumps the next	Fairly good
mental especially when head uncovered	Depression	None	—	—	Fairly good
frontal region when bending and on exertion	Depression	None	Light work	—	Fairly good
reaction of operation excitement and on exertion	Insomnia	One a month	Delivering letters three hours	—	Fairly good
		Chauffeur	—	—	Good

Continued on next page

Table II—LIST OF 40 DECOMPRESSION CASES

DATE AND NATURE OF INJURY	SYMPTOMS	OPERATION AND DATE	PRESIDENT STATE AS TO HEADACHES	DID OPERATION BRING RELIEF?
G S W Frontal, fissured, July 1915 Returned to France headaches commencing May 1918 and gradually getting worse	Persistent headaches	Left subtemporal decompression 24/3/19 Very marked oedema	Frequent headaches though better	' Still bad at times '
G S W Left frontal, penetrating 26/4/17	Headaches varying from slight to awful	Left subtemporal 24/3/19 Some oedema and some bulging	Occasional	—
Fractured base, 21/12/18	Headaches Dull mentality	Left subtemporal 1/5/19 Moderate degree of oedema	Occasional	—
Concussion after mule kick occipital, 10/4/19	Constant headaches dull and apathetic	Left subtemporal 29/5/19 Very marked oedema	Slight occasional	Still improving weaker fully would hardly know me for the same man'
Epilepsy since 7 years aggravated by service	Frequent fits severe headaches both becoming worse	Left subtemporal decompression, 30/4/19 Marked oedema	No headaches	Yes marked
G S W Left occipital, penetrating FB removed from right parietal region, followed by hernia cerebri	Very severe headaches, general convulsions papill oedema Streptococci in cerebrospinal fluid	Left subtemporal decompression 15/3/19 Little oedema	Constant headaches	Little if any
Blown up concussion 26/1/15	Constant headaches with numerous epileptiform fits	Left subtemporal decompression 20/6/19 Moderate degree of oedema	Occasional slight headaches	Yes
G S W Left parieto occipital May 1916	Severe headaches dulled brain ventriculitis and right hemiplegia	Left subtemporal decompression 13/12/19 Much bulging and little oedema	Frequent and severe	—
Concussion March, 1919	Very severe headaches apathetic and depressed	Left subtemporal 20/11/19 Very marked oedema Right subtemporal 18/12/19 less oedema	Occasional slight to moderate	Yes and improving
G S W Left parieto frontal penetrating 12/4/18	Severe and persistent headaches with exacerbations dull mentality	Left subtemporal decompression 31/5/18 Marked oedema	Occasional	Yes much better
G S W Left frontal non penetrating 15/5/18	Frequent headaches dull and apathetic	Left subtemporal decompression 27/10/19 Some oedema some bulging	Very slight	Yes and still improving

IX CLOSURE OR PROTECTION OF THE GAP IN THE SKULL

In discussing this question two main points arise (I) *What is the object of the procedure what is it done for?* (II) *What is the best method of closing or protecting the aperture in the skull?*

1 The Objects of the Procedure—These are three in number—

1 To relieve the psychological effects produced on the patient by the mere fact that he has a hole in his skull Can they be remedied by operation?

2 To afford some means of protection of the gap from further external injury

WITH PARTICULARS AND END-RESULTS—*continued*

WHEN AND WHERE ARE THE HEADACHES?	ANY OTHER COMPLAINTS?	ANY FITS?	WORKING?	REMARKS FROM PATIENTS	CONCLUSIONS
In region of operation	None	None	—	—	Moderate only
Any exertion	None	None	Odd man at farm work	—	Fair
—	None	None	—	—	Fair
—	None	None	Light farm work	—	Good
—	—	Occasional lasting 2 minutes	Light gardening	'Much better since my operation. The fits do not worry me as of old and they are less frequent, and not so severe.'	Good
Cerebral	Weakness of left side	Occasional epileptiform	—	'Very little better, life saved only.'	Bad
Over eyes	—	None since operation	Traveller in cloth	'You found out about my head more in a few hours than what it took many doctors months—and also removed my trouble, what the others could not do.'	Good
Fronto occipital	—	Occasional slight	House work	'Hope to go to Canada soon.'	Fair
—	—	—	—	—	—
Frontal	—	—	Attendant at a London hospital	Never get those terrors of headaches and I should not be writing this except for you.	Good
Frontal	—	—	Tennis racket stringing	Am very satisfied	Good

1 To relieve the patient of certain symptoms associated with and dependent on the injury more especially headaches fits and paralyses.

1 *Psychological* The mere fact that a man has a 'hole in his skull' may bring about marked psychological effects more especially when the defect is of considerable size situated over the more conspicuous parts of the head covered by a scar overlying a pulsating brain and depressed below the surface contour of the skull—as so many of these injuries are.

The patient may feel the defect both literally and metaphorically worrying himself about it, lingering it, and imagining all sorts of terrible after-results. Such defects render

the patient supersensitive to observation, and he may be desirous of operation on purely psychological grounds

Such patients form but a small group, but it is possible that an operation so planned as to render the deformity less obvious may bring about quite satisfactory results, more especially when the injury is situated in the frontal region. Silver and celluloid plates applied in the manner described below, may benefit the patient, but all such methods present one obvious disadvantage—there is unquestionably a definite tendency for all such plates to assume, as time progresses, a degree of concavity to the surface, the result of atmospheric pressure—there is, as a rule, a shrinkage of the brain in immediate relation to the defect of the skull, and the plate gives to the external pressure, bending inwards towards the brain. This results in a concavity of the plate, and frustrates the object desired. Bone-plates will be more satisfactory in this class of case, and I regard the purely psychological cases as the only indication for bone-grafts.

In other words, when the patient is influenced solely by psychological effects, the aperture may be closed in, if otherwise desirable, with a bone-graft. All other methods are liable to fail, with corresponding disappointment to the patient.

2 *Protective*—When a patient with a gap in his skull suffers from nothing more than a fear of possible injury, some form of plating may be desirable to strengthen the injured region. These cases are very few in number, and I do not remember more than two or three coming to me with that specific complaint. In such cases I think the double celluloid plate method will suffice, on the ground that no method, even bone-grafting, is really protective against any serious direct injury to the region, and the celluloid method is sufficiently efficacious and quite simple.

3 *Symptomatic*—The more important remote effects of gunshot wounds of the head, where a defect in the skull remains, may, so far as symptoms are concerned, be divided into two groups, a major group where the injury is followed by severe, often persistent headaches, fits, and paralyses, and a minor group where irritability, insomnia, giddiness, and nervousness are predominant. It having been stated elsewhere that closing in or protecting the gap in the skull may improve the conditions specified, it is necessary to examine the question carefully, in the endeavour to arrive at some definite conclusion as to the degree, if any, to which the patients have benefited.

Firstly, I would ask these questions. Can any of the conditions enumerated be regarded as dependent, in their incidence and progress, on the mere presence of some osseous defect? At first sight, it would appear highly improbable that any of these symptoms could be explained on this hypothesis, and that they could be remedied by closure or protection of the aperture. There is, I think, but little doubt that that is a correct representation of the case. But there are some few cases in which operation is really beneficial. There are some patients who suffer from slight infrequent headaches, who sleep badly, who suffer from very slight epileptiform or fainting attacks, who are depressed and nervous—cases, in other words, of the mildest forms of after-effect. The closure or protection of the aperture of the skull, in such cases, often brings about great benefit—though it is possible, according to my mind, that this is mainly due to the psychological effects of the operation.

In consideration of the more serious symptoms, severe headaches, etc., the headaches are due, as explained previously, to a condition of cerebral oedema, for which decompression is indicated as the rational treatment—not the closure or protection of the skull defect.

The various paralyses are due to tract degeneration, and plating, etc., cannot lessen the degree and extent of the paralyses. The fits, too, are as a rule secondary to cortical scarring and degeneration, and gap protection cannot lower their incidence. Nervousness, insomnia, giddiness, etc., are not of psychological origin, they form part of the syndrome of cerebral oedema, and are due, I think, to the soaked sodden state of the cerebral cortex. At first sight, therefore, one would be inclined to accept the view that closure or protection of the gap in the skull could not bring about any appreciable benefit along the lines indicated.

But the question is not quite so simple. Fits, for example, may be brought about by adhesion of the brain to the overlying scalp scar, and it is clear that the interposition of plates (*see below*) between the scalp and the bone, merely separating brain from scalp is not infrequently beneficial, sometimes markedly so.

Again I would lay considerable stress on Sargent's theory—that in penetrating wounds of the brain, the dura mater becomes adherent to the margins of the osseous gap, and that the brain is adherent in the immediate vicinity of the site of dural injury to the dura itself, and, in consequence, that the brain is more or less 'anchored' to the region of the scalp and bone injury. There is under normal conditions a certain degree of mass movement of the head and any 'anchoring' of the brain may assist the brain to rapid movements of the head and any spreading oedema, in the development of fits by local irritation in the advent and persistence of localized and general headaches by reason of the local and spreading referred to later, which has as a definite object the freeing of the adherent dura the 'un-anchoring' of the brain (I apolo- theories, Sargent has advised a method of plating referred to later, which has as a give for the use of this term), and the prevention of further adhesion in that region. It would appear therefore, that there are some points to be advanced in favour of closure or protection of the gap in the skull from the point of view of relief of some of the remote symptoms. Acting on Sargent's theories I have plated defects on many occasions with tiges and disintegrated this method. Further details will be given later as to the advan-

1 Taking the question, then is a whole one would be inclined to conclude —
 1 That in the event of severe headaches with exacerbations and prostrations cerebral oedema being in most cases the causative agent, plating is useless. Decompres-

2 That, in the event of hemiplegia or paralysis of corresponding magnitude plating brings about no benefit.
 3 That in the event of severe Jacksonian fits of epileptic or epileptiform fits little benefit is likely to accrue from closure or protection of the gap with bone-plates or other plates.

But when the headaches are mild and inconstant more especially when limited to the immediate vicinity of the wound when the fits are slight, generalized and slight or Jacksonian and slight, and when one or both of these conditions are associated with monomania, trepidation and giddiness then I believe that with plating or closure of the defect along the lines suggested by Sargent there is a fair probability of amelioration.

The plating question has to be considered most carefully. I have seen cases which have been plated both by myself and other surgeons where although the immediate results were satisfactory after a few months the old troubles returned sometimes more forcibly than previous to the plating. And I have removed the plates in some few of the cases with immediate relief of all symptoms. In these cases where plate removal was necessitated the patients had complained of a great feeling of weight of oppression in the region of the wound with vague neuralgic pains radiating from the plate but usually centred at some part of its circumference often in the line and distribution of some sensory scalp nerve.

In general my present opinion on plating of all types so far as indications and results are concerned is as follows:
 1 From the point of view of psychological effects operation closure or protection of the aperture is of some but limited benefit.
 2 From the point of view of symptomatic relief the operation may be of definite value when carried out in the type of case indicated but is disappointing in general more especially when conducted for the relief of severe and more or less constant headache of fit cases of the more severe type whether generalized or Jacksonian and of paroxysms.

3 From the point of view of protection the operation is of considerable benefit when carried out in the type of case indicated but is disappointing in general more especially when conducted for the relief of severe and more or less constant headache of fit cases of the more severe type whether generalized or Jacksonian and of paroxysms.

II What is the Best Method for Closing or Protecting a Gap in the Skull ?—

The various methods that have been adopted, both previous to the war and subsequently, may be divided into two groups (1) *Autogenous bone-grafts*, to close permanently the gap in the skull (2) *Plating of the defect*, together with other procedures on the lines of Sargent's theories (Fig 107)

I BONE-GRAFTS—I have stated previously that I do not like bone-grafts for the skull I will admit at once that I have not used this method myself, and therefore my

opinions are not based on practical experience, but I have considered their advantages and disadvantages, their merits in a general sense, and I have seen about a dozen cases in which this method of skull protection has been carried out I do not like the principle, nor do I like the results as seen in these few cases

So far as principles are concerned, I have already pointed out that, so far as my personal experience goes, the benefit attained by closure or protection of the aperture in the skull by any method is very limited in practice, that it ought not to be adopted with the idea that any considerable improvement in respect to fits, headache, and paralyses, is likely to accrue and I would add further that in my opinion, the complete closure of the gap with a bone-graft which is expected permanently to close in the defect is wrong in principle It may be useful when the gap alone is the trouble, where the patient is perfectly well except for the hole in his skull, but such cases are relatively few in number, almost negligibly so the great majority suffer, to a greater or less degree, from those other remote effects mentioned previously, all due to intradural changes, whether excess fluid adhesions or degener-

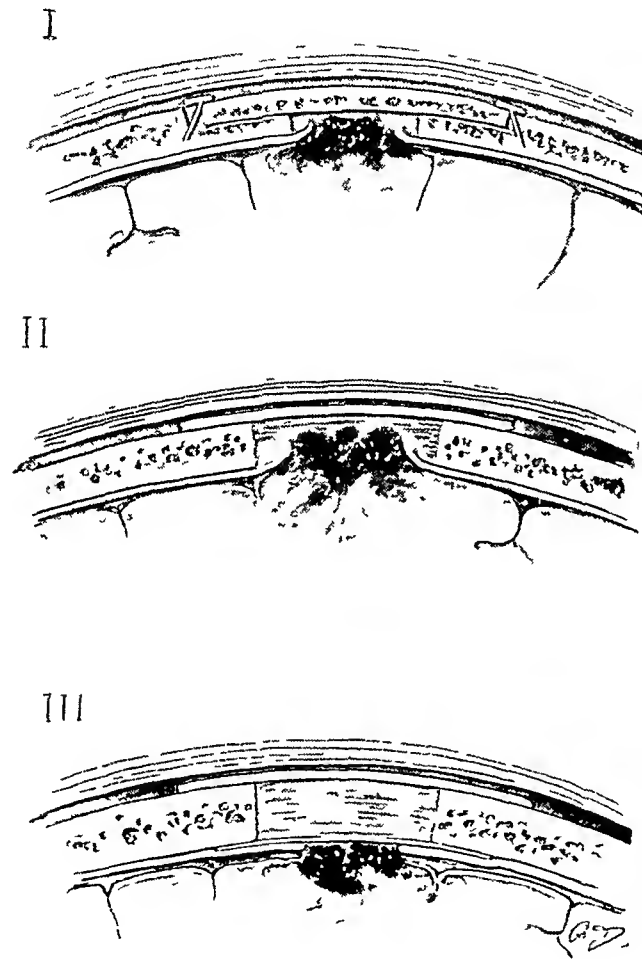


FIG 107—To illustrate three methods of protection or closure of defects in the skull (i) By bone-graft (ii) By silver plate between scalp and bone (iii) By two celluloid plates—an inner thin smooth plate between bone and dura an outer perforated and stronger plate between scalp and bone

erations To pay all attention to permanent closure of the gap and to neglect the far more important intracranial changes—to dam up excess fluid, for example—is, in my opinion, entirely wrong in principle

In further reference to bone-grafts, I have observed, in the cases that have come under my care that the results obtained have been poor Further, I am not convinced that the grafts will remain as bone-grafts—the skull is a poor place for bone growth in general and in two or three cases that I have seen, the graft died leaving a plate of dead

bone exposed to the surface through sinuses discharging pus—the graft being removed subsequently with considerable difficulty

My arguments are undoubtedly weakened by the fact that I have no actual experience of bone grafting in the skull, but I believe I am right in principle and practice

2 PLATING OF THE DEFECT—Following Sargent in his theories and practice, I am accustomed to adopt the following procedures—described as briefly as possible

Material used—Celluloid plates two in number, the one (the outer plate) perforated, $\frac{1}{800}$ in thick, the other (the inner plate) smooth, $\frac{1}{1000}$ in thick. These plates are bought in sheets and can be cut readily to the size desired

Sterilization of the Plate—The fresh celluloid, cut to a convenient size and shape (Fig 108), is washed in running water, scrubbed with soft soap and water rinsed again in running water, then wiped over thoroughly with methylated spirit, wrapped in sterilized gauze, and put away till wanted. When required,

the celluloid is placed in sterilized water, and previous to insertion is rinsed in spirit then

washed again, after which it can be used. Immediately previous to insertion, the plate is picked up with forceps and cut to the size and shape required

The thicker perforated plate will lie between the bone and the scalp—protective—whilst the smooth plate will be used between the dura mater and the bone, after the un-anchoring of the dura mater from the margins of the aperture

Method of Introduction—

Under general anaesthesia—the head being enveloped in gauze sheet to avoid contact with the patient's skin—a flap is turned down, the whole thickness of the scalp, completely exposing the defect in the skull. This scalp incision lies one-eighth to three-quarters of an inch distal to the margins of the aperture in the

bone. Some part of the scalp flap may be merely scar-tissue, adherent to the tissue immediately underlying the gap, and great care must be taken, when turning down the

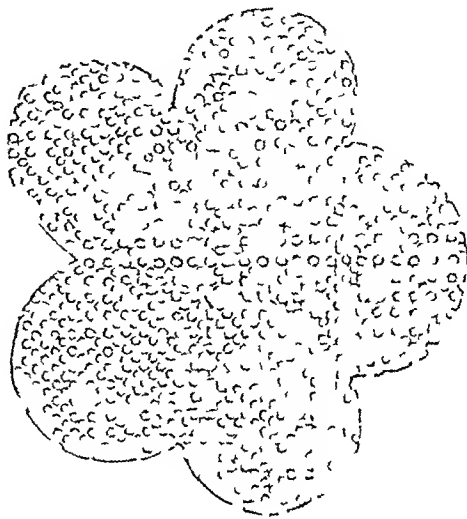


FIG 108—Closure or protection of apertures in the skull. The thicker outer perforated celluloid plate for insertion between scalp and bone. This plate is trimmed to some such shape as is represented in the illustration, for ready insertion underneath the scalp.

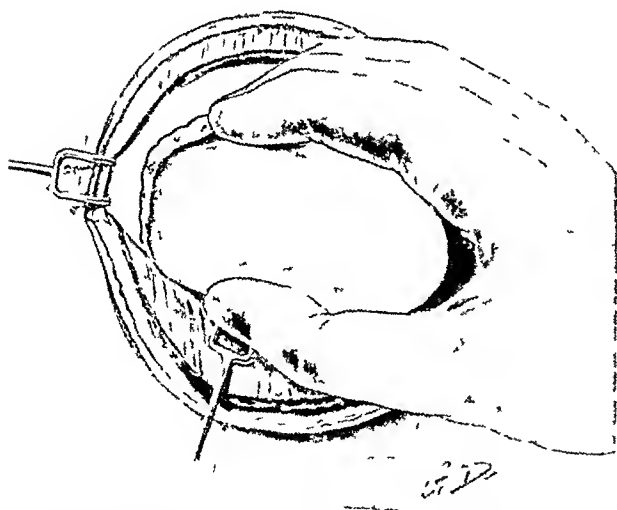


FIG 109—Closure or protection of apertures in the skull. The introduction of the inner thin celluloid plate, in insertion of the plate between the dura and the bone edge.

scalp flap, to avoid button-holing the flap. All surface tissue must be included in the scalp-flap by clean dissection—even at the expense of the underlying dura, torn dura, or scar-tissue—this should be avoided whenever possible. Bleeding is controlled with the scalp tourniquet, or by means of hemostatic forceps.

Insertion of the Inner Plate—The pericranium is incised with a fresh scalpel, about half an inch distal to the margin of the gap, and stripped with Farabeuf's raspator to the margin of the aperture. There it becomes adherent to the edge of the bone, to which also the dura mater is adherent in the case of a penetrating wound. With the raspator insinuated around and beneath the osseous deficiency, the dura mater and pericranial fringe are detached from the margins of the gap, after which a flat periosteal elevator is inserted so as to strip the dura from the overlying bone for a distance of not less than one inch throughout the whole circumference of the gap. Thus being completed, the ragged tissue in the centre of the field is dissected away with a sharp scalpel, leaving a surface as smooth as possible, without cutting into the surface of the cortex exposed. It is important in every case to avoid injury to cortical vessels, venous sinuses, etc. This must be carried out with circumspection, a dry field remaining.

All is now ready for the insertion of the inner, smooth plate, between the bone and the dura mater (*Fig 109*). If, however, the bone edges are ragged they should be smoothed with nibbling forceps, it being the general object to leave an oval opening in the bone—the plates are then introduced with ease. The thin celluloid is trimmed with scissors to correspond to the size and shape of the aperture, being about one third of an inch larger in all diameters. It is bent or doubled so as to slide and be inserted beneath the bone, lying there snugly, overlapped by the margins of the gap throughout its circumference.

Insertion of the Outer Plate—The scalp having been separated from the bone for a distance of about two inches peripheral to the margins of the gap, the thicker celluloid is trimmed with the scissors, with such snips here and there as will allow of the snug fitting of the plate to the convexity of the skull. The plate should overlap the margin of the aperture in the skull by not less than one inch, the edges of the plate being guided beneath the scalp—so as to overlie the gap and be overlapped by the scalp. When lying smoothly the scalp flap is replaced and anchored by many fine salmon-gut sutures, without drainage. Dressings and bandages are applied carefully, with the object of exercising an equal pressure throughout the wound area, thus avoiding the development of a hematoma.

Having been compelled to remove celluloid plates on some few occasions, twice for sepsis, and a few times because of recurrence of headache, etc., it is interesting to note how the two plates work. With respect to the septic cases, I would urge that if the temperature of the patient after operation suggests that possibility, and when the wound, on inspection, suggests the presence of a hematoma, it is wise to act promptly, turning down the flap and removing both plates. This is readily effected in the early stages. In one case, blood external to the outer plate was sterile, whilst the blood between the two plates grew pyogenic organisms. In the other case, nothing grew, but the hematoma was of considerable size, and I think I acted judiciously in removing both the plates.

When the plates are removed at a later date—months after the insertion—the following conditions were found. The outer plate was firmly anchored in position (being removed indeed with some difficulty) by strands of fibrous tissue, which had passed through all the small perforations of the plate, from scalp to bone and pericranium, except over the immediate region of the aperture itself, where the strands passed from the scalp into the perforations of the plate, but when the plate was removed and looked at from the under surface it could be seen that where the outer plate had come into 'approximation' with the inner plate it was smooth, lined with an endothelial membrane, and no strands of fibrous tissue had here passed through the perforations.

The inner plate was equally interesting. It fulfilled its purpose admirably, that is (1) It was non-irritating, as proved by the fact that after having been in position for some months, it could be lifted off from the surface of the brain or dura with the greatest ease with no sign whatsoever of fibrous tissue development, and (2) It had prevented

'anchoring' of the brain—that is to say, adherence of the dura mater to the margins of the aperture. This was so. The 'un-anchoring' process was complete and apparently permanent.

DISADVANTAGES OF CELLULOID PLATES—

Special Disadvantages—The inward sinking or depression of celluloid plates, seen in some cases more especially those associated with considerable primary brain laceration, might be lessened by the utilization of an outer silver plate, but the silver plates are rather more bulky, less readily adapted to the curvature of the skull in the region of the defect, and less comfortable to the patient. Silver, gold, and aluminium plates are also inclined to yield to the atmospheric pressure and to become depressed. They could, of course, be made so thick as to obviate such secondary changes, but they then become bulky and generally uncomfortable.

General Disadvantages—If the plating is carried out along the lines indicated, these celluloid plates are, in general, fairly satisfactory. For the relief of headaches, fits, and paralyses, they are—as are all plates—useless or harmful. Celluloid plates are readily sterilized, if harmful, they can be removed easily at an early date—with more difficulty later. This is one of the great advantages of these plates over all other methods, more especially over bone-grafts.

From the point of view of protection from further injury, many of my patients are advised to wear thin aluminium shields, covered with cloth and fastening round the head with narrow elastic. Some patients object to these shields because of the attention which is directed towards their head trouble. I point out to them that the shield should be regarded as a shield of honour.

X POSTSCRIPTUM

Since this paper was written as a careful and considered resume of the remote effects of gunshot wounds of the head, I have been rather perturbed by some recent fatal cases. Before alluding to these disturbing factors, it should be noted that, of 775 fully-recorded cases, 50 died (6.5 per cent). Death occurred, on an average, nine weeks from the date of the primary injury. With one exception, where death resulted from tetanus, the fatal termination was due to meningo-encephalitis. In no case did the autopsy reveal a localized abscess—that is to say, an abscess with well-defined boundary, the abscess, if present, was often loculated and of considerable size, spreading towards the surface of the brain or involving the ventricles.

In 19 of these fatal cases (38 per cent) foreign bodies were present, too deeply situated for removal, and in 22 (44 per cent) the conditions were complicated by the presence of a hernia of the cerebrum or cerebellum. In 12 cases (24 per cent) the lesion was situated in the occipito cerebellar region.

It would appear probable that, in the 19 cases where foreign bodies were present, the foreign body in itself was not responsible for the death of the patient—42 cases are discussed elsewhere in this article with foreign bodies remaining in the brain, some in the most inaccessible positions, who are alive and well, and 58 per cent of whom are at work of some description. It is noteworthy also that, although 22 cases died with hernia cerebri, yet 35 cases recovered, 30 per cent being at work at the present time.

Again, the fatal termination of 19 cases with foreign bodies retained must in no sense be taken as an argument for more radical early efforts at removal of a foreign body. From a general survey of such cases I think it is clear that the surgeons in France did not err on the side of leniency in their attempts at foreign body removal—whether the opposite was the case, I am unable to offer an opinion.

Although the average duration of life, from the date of injury till death occurred, was about nine weeks, one case lived eight and a half months, being discharged from hospital with a healed wound, and re-admitted two months later with breaking-down wound and hernia cerebri. This case has some bearing on the more recent perturbing cases alluded to at the beginning of this postscriptum.

The more prolonged of the fatal cases gave warning as to their general unsatisfactory condition by (1) persistent headaches, (2) bouts of vomiting, (3) great depression and irritability, and (4) steady emaciation. In some cases the conditions were diagnosed long before death by means of lumbar puncture, and, in the event of positive result, more energetic treatment was at once carried out—repeated lumbar puncture, subtemporal decompression, attempts at the removal of the foreign body, drainage of abscess, vaccines, etc. I was always rather afraid of lumbar puncture—the risk of spreading the infection is so obvious—but in some cases this method of treatment was carried out energetically.

Even when meningo-encephalitis is existent, recovery may still take place, as in the following case of a left temporo-occipital penetrating gunshot wound, with numerous small metallic and osseous fragments in the underlying brain substance, violent attacks of headache, and screaming fits. Streptococci were found in the cerebrospinal fluid on all occasions when lumbar puncture was carried out. Treatment—repeated lumbar puncture and subtemporal decompression for the relief of headaches. Patient recovered and is now seeking to obtain employment. There were some other cases of similar nature.

Now, as to the perturbing factors. Within the last six months I have had under my care, or have been asked to see, four cases where old wounds had ‘flared’ after having been healed for three to five years—in two cases the men had been doing ordinary work for this time, with no serious disability other than periodic headaches. Then the wound ‘flared’ just as so many old wounds of the extremities do, the patient rapidly became unconscious, with epileptiform fits, and died, in spite of immediate radical treatment. In three of these cases the autopsy showed meningo-encephalitis, with no foreign bodies retained, and in the fourth case, a non-penetrating wound of the frontal region, there was an old hemorrhage in the subjacent frontal lobe, with recent extension, and death ensued from status epilepticus.

These four cases known to me—and there are probably many others of similar nature—lead one to think of the many patients suffering from the effects of gunshot wounds of the head, many of whom have foreign bodies retained in the brain substance. What will be their subsequent history?

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THE EARLY SIGNS AND SYMPTOMS OF CHOLELITHIASIS

By SIR BERKELEY MOYNIHAN, BART, KCMG, CB, IRLES

It has probably been the experience of many surgeons to operate upon cases in which a diagnosis of cholelithiasis has been made, and to fail to find any stones within the gall-bladder.

In such cases many years ago, I was content to drain the gall-bladder, and I found in a disturbing number of occasions that the bile was sterile. The gall-bladder looked normal in many of them, but many presented those early signs of disease which I now recognize at a glance. In the cases which had been drained, a temporary abeyance of symptoms was almost constantly observed, but a recurrence rarely failed. A second operation was performed and the gall-bladder removed. In 1909 I described "A Disease of the Gall-bladder requiring Cholecystectomy," a disease unrelieved by cholecystotomy, in which the gall-bladder wall itself presented the evidences of chronic or subacute inflammation. There was a denudation or destruction of the villi, with a deposit of lipid material, especially cholesterol esters, in the stroma of the mucous membrane. To this condition MacCarthy later gave the very appropriate descriptive name of the strawberry gall-bladder. The appearance of the living membrane exactly resembles that of a ripe strawberry, the congested mucosa being studded with brighter yellow dots which end abruptly at the cystic duct.

There are more types of 'strawberry gall-bladder' than one, and the differences probably represent stages of gradual development. In its earliest, but quite definite, form, the mucosa is a little redder than the normal, and the slightly yellow specks show nothing but lipid material. It is possible, as Professor M. Stewart has suggested to me, that in this stage the gall-bladder merely represents a local phase of the general condition hypercholesterolemia, and that infection has not yet developed in its walls. In later stages the villi may become denuded of epithelium, and tiny ulcers soon develop upon the surface, or cholesterol crystals, like fine grains of sand, may firmly adhere to, or be embedded in, its walls. In the latest stage the gall-bladder becomes thickened throughout, a firm and copious deposit of fibrous tissue is found in its walls, and calculi are often present.

This experience led to a closer study of the gall-bladder walls, and of the bacterial content of the bile in cases in which cholelithiasis was present, or was suspected, and, as more and more cases have come under review, it has by degrees become clear that there are conditions of the gall-bladder, apart from calculous disease, which cause a close mimicry of the symptoms of gall-stones, and which can be successfully treated only by cholecystectomy. The diagnoses we make of abdominal diseases are often inferences only, and not certainties, however much we may be tempted so to regard them. If a patient suffers from repeated attacks of pain in the upper abdomen associated with a rigor, which is indicated upon the 'steeples' temperature chart, if jaundice, which is always present, deepens after the attack and gradually subsides until the next attack, and if there is a progressive loss of weight, we do not hesitate to diagnose a floating stone in the common bile-duct. But the symptoms are not those of stone, but those of a cholangitis, which may be and usually is provoked by a stone, but which may be provoked by other conditions also, such as a series of hydatid cysts escaping down from the liver (as I have seen twice), or a pineal calculus in the ampulla, or a subacute or chronic pancreatitis. So it is I think, with the diagnosis of stones in the gall-bladder. The condition that provokes the symptoms is an infection of the gall-bladder, set up it may be by stones, but not seldom existing in an early or advanced degree in the absence of stones. The

whole course of these diseases of the gall-bladder or of the liver, associated with gall-stones, is not yet by any means clear to us, but our knowledge is widening little by little, and a broad conception of the whole problem is now possible.

By what means and through what channels is the gall-bladder infected?

There are several possible avenues which may be traversed by invading micro organisms

1 INFECTION MAY ASCEND FROM THE DUODENUM, along the common and cystic ducts to the gall-bladder, or along the common and hepatic ducts to the liver—This mode of infection, if it exists at all, is probably very rare. Bond's experiments have shown that pigments introduced into the rectum can soon be recognized in the discharge from the gall-bladder after cholecystotomy. It is certain, therefore, that organisms can travel directly upwards in these reflux currents. But they probably do not, because the duodenum is as a rule, sterile, and is very rarely infected heavily. The downward current of bile flushes the common duct with a certain regularity.

2 INFECTION MAY DESCEND FROM THE LIVER—Organisms reach the liver by way of the portal stream. As the blood passes round the liver lobules its organisms are caught up by the hepatic cells, which are the great 'destructors', and are rendered inert or killed. Some few may escape with their lives, perhaps at a time when the liver is momentarily overwhelmed by large numbers of organisms. Those which so escape gain access to the gall-bladder, and may form the nucleus of stones, which make haste to develop round them. The portal blood consists of two main streams, one from the alimentary canal and one from the spleen. The view has been generally held that the former is the current along which most of the micro organisms travel, and this no doubt is true. But remembrance should also be given to the possibility that organisms may be derived from the spleen. The association of diseases of the liver, and of gall-stones, with diseases that seem to have their origin or their chief development in the spleen has recently become clearer. In cases of hæmolytic jaundice, 60 per cent of the patients suffer also from cholelithiasis, with splenic anæmia, cirrhosis of the liver and gall stones are both associated. Enlargement of the spleen is noticed in cases of stones in the gall-bladder and the duct, but sufficient regard has not been paid to the possibility that it is from the spleen that the infective agent is immediately derived. There are cases in which a large number of small stones are found throughout the substance of the liver, not only in cases of cirrhosis, but in cases where the liver appears little if at all changed from the normal. And every surgeon is familiar with cases of recurrent gall-stones in which the common duct and all the ducts of the liver within reach are filled with mud and fine stones, which may be washed down in almost unending quantities. In such cases I pass several tubes up into the liver, and apply the Carrel method of intermittent irrigation for several weeks. About ten months ago I operated on such a case in which seven operations had been performed. I dealt with the bile-ducts as I have described, and then removed a spleen that was enlarged to approximately three the normal size. Since that time no attacks of pain or jaundice have returned, and as this is by far the longest interval of freedom the patient has had for some years, I am hoping that we may have cut off the source of supply of the infecting organisms to the liver. Splenectomy for recurrent cholelithiasis may be found necessary in similar cases. One of the functions of the spleen is to filter out micro organisms and toxic substances from the blood-stream, and to send them to the liver for destruction. It may sometimes harbour them, rather than transmit them. Its capacity to do so in syphilis has been shown by W. J. Mayo. Possibly in other infectious micro-organisms or toxic materials are held up and passed on only from time to time to the liver, which in this way receives the material upon which gall stones are deposited.

3 INFECTION MAY BE DERIVED FROM THE BLOOD—We owe our knowledge on this subject to Rosenow.¹ He found that organisms removed from the gall-bladder, from the bile from the centre of gall-stones or from the cystic gland of patients treated by cholecystectomy contained organisms, chiefly streptococci, which when injected intravenously into animals produced lesions of the gall bladder, the bile-ducts and some-

times of the stomach or duodenum. He suggested that such organisms have in 'elective affinity' for the tissues of the like kind to those from which they were originally derived. Such organisms reach the gall-bladder of the animal by the blood-stream, and in the gall bladder produce lesions exactly comparable to those in the organs from which they were taken. Whether it is the micro-organism that selects the tissue in this elective affinity, or whether it is the soil that alone provides the culture medium necessary for the growth of the germs which are scattered everywhere in the blood-stream—the soil selecting the germ—is not a matter of importance. The truth is well established by Rose-now's experiments and by clinical and pathological research in man, that micro-organisms attacking the gall-bladder may reach it through the blood-stream.

The question has been most closely studied in connection with typhoid fever, but the results of the experimental work appear very conflicting. J. Koch² in a patient who had died of enteric fever, found inflammatory changes in the mucous and submucous layers of the gall-bladder. Just beneath the epithelial layer of the villi he found masses or clumps of organisms, apparently those of typhoid fever. No organisms were found on the surface of the mucosa. He therefore drew the conclusion that it was not from the bile that the gall-bladder was infected but by a process of embolism. In the nests of organisms in the wall of the gall-bladder propagation took place, organisms being liberated and escaping through the mucosa into the gall-bladder to infect the bile. Chiarolanza³ injected typhoid bacilli into the veins and beneath the skin of rabbits and described the organisms as forming emboli in the capillaries of the submucous layer of the folds of the gall bladder. Other observers, among them Grode, have however recovered organisms injected into the veins from the bile descending from the liver.

The investigations of Gosset, Loevy and Magrow⁴ show that calculi may originate inside the villi of the mucous membrane as minute collections of cells surrounded by cholesterol. As they grow they detach themselves from the wall of the gall-bladder, and becoming free within its cavity, they increase in size, and press upon each other until they become freed. In any large collection of stones in the gall-bladder two or more generations may be recognized groups erected in the same period of infection being of almost equal size—the larger the stones the longer their existence. The conveyance of organisms by the blood-stream to the gall-bladder probably accounts for those cases (examples are not very infrequent) in which an acute cholecystitis or appendicitis follows rapidly upon such infections as tonsillitis and influenza, or pancreatitis or orchitis upon an attack of mumps.

4. INFECTION MAY REACH THE GALL-BLADDER FROM THE LIVER BY WAY OF THE LYMPHATICS.—The lymphatics of the gall-bladder communicate freely with those of the liver. Affections of the liver, changes in size and changes in the cells have been noticed very irregularly by most surgeons. It would be well if a note of the size and condition of the liver could be embodied in all accounts of operations for gall-stones. If, along with the gall-bladder, a piece of the liver is removed, it should be submitted to microscopic examination. E. A. Graham⁵ noticed in a series of 30 cases that the liver was enlarged in 26. In the remaining four there was definite gross evidence of a previous or existing pathological change in the liver other than an enlargement. Inflammatory changes, chiefly of the nature of pericholangitis were constantly observed in cases of acute and subacute cholecystitis. Graham suggests that an involvement of the liver is "so frequently in accompaniment of cholecystitis that the association must be practically a constant one."

Sidler⁶ has shown the intimate connection which exists between the surface lymphatics of the liver and the lymphatics of the gall-bladder through the attachments of the latter to the fossa in which it lies. The view is held that it is through these lymphatics that the gall bladder may be infected from the liver, that cholecystitis is secondary to hepatitis. My own experience gives support to this hypothesis. Gross affections of the liver which could conceivably be regarded as antecedent to the gall-bladder infections found at operation are present in less than one-fourth of the total number of cases submitted to operation, and among these must be included all those cases where a splenic

condition could have been responsible for the hepatic enlargement or disease, and those in which these conditions were probably secondary to the gall-bladder disease. But the history of attacks in which enlargement of the liver has temporarily occurred (a sort of œdema or phlegmon) is occasionally to be obtained. In the examination of specimens by the microscope cases are seen in which the peritoneal and subperitoneal coats are invaded by infection when the mucous and submucous coats are normal. In these cases infection must reach the gall-bladder either by the lymphatics, which is most probable or possibly by the blood-vessels. When the infection arrives through the blood-vessels it is the submucosa that is first affected in almost every instance.

The view has also been taken that the pancreatic inflammations which are found associated with cholelithiasis are due to a pancreatic lymphangitis. It is difficult to say with certainty how often the pancreas is affected in cases of cholelithiasis. Conditions such as swelling of the head of the pancreas, or hardening or fibrosis, are very difficult to assess, and mere palpation exposes an opinion based upon it to many errors. My estimate is conservative one, I think, places the frequency of pancreatic implication in cholelithiasis at 12 per cent. The removal of a tiny portion of the pancreas gives valuable information but it is not as often practised as it might be. Thuroloz and others have suggested that the free communications of the lymphatics of the gall-bladder and the bile ducts with those of the pancreas, the whole forming one plexus, explain the origin of pancreatic inflammation secondary to cholecystitis and cholangitis, and they discredit the previously accepted view that the infection travels by way of the cystic and common ducts. And Deaver has added the weight of his great authority to this teaching. He writes that 'most cases classed together under the general term of chronic pancreatitis are at first really cases of pancreatic lymphangitis, the infection being propagated from the gall-bladder and bile-ducts or from the pyloric region of the intestine along their efferent lymph channels, which come into intimate relation with those surrounding and embedded in the head of the pancreas.'

We do not know, however, that the infection of the pancreas usually spreads from its surface inwards rather than from the duct outwards to the body of the gland.

It is true that in cholecystitis the cystic gland is always enlarged, and that in cholangitis the glands along the duct may be so large and so hard as to make the discrimination between them and stones very difficult. In such cases the supra-pancreatic glands may also be enlarged. Nordmann's experiments seem, however, to controvert the view that invasion of the pancreas is primarily lymphatic. If in the dog a ligature is placed around the opening of the ampulla of Vater into the duodenum, the common bile duct and the upper duct of the pancreas are then directly continuous one with the other. If, after this ligature, a virulent culture is introduced into the gall-bladder, acute pancreatitis develops. If the same culture is introduced and the cystic duct at once ligatured, no pancreatitis develops. In these experiments at least the conveyance of the infection from the gall-bladder to the pancreas is by the way of the ducts, and not through the lymphatics. And probably this is often if not generally true of the acute condition in man also.

5 INJECTION MAY REACH THE GALL-BLADDER BY DIRECT CONTINUITY.—This method is rare. Gastric and duodenal ulcers—especially the latter—may have the gall bladder adherent to them. The duodenum is sometimes saved from perforation by having the gall-bladder soldered on to its outer surface. I have on many occasions found an inflamed appendix either adherent to the gall-bladder or in closest contiguity to it. Infection may penetrate the gall-bladder from its serous surface inwards in such cases, but in the aggregate they may be very few in number and from the point of view of the development of gall-stones they are negligible.

The examination of a large number of gall-bladders shows that infection begins with almost equal frequency on the mucous surface and on the peritoneal coat. From the mucosa it penetrates by degrees deeper and deeper until the elastic coat has disappeared and the muscular coats are at last destroyed. An interesting observation that we have made shows that even an early invasion of the submucosa is often indicated by the develop-

ment beneath the peritoneum of the gall-bladder of a considerable deposit of fat. It would seem as though a warning had reached the serous covering that it must protect the general peritoneal cavity from the impending perforation of the coats of the gall-bladder. A fat deposit fulfilling the like purpose is often seen elsewhere. A gastric nodule lying on the lesser curvature has often a large mass of fat developed around it, a septic kidney is swathed in thick masses of fat, a chronically infected appendix has a grossly thickened mesentery, diverticula of the left colon are covered with fat, and so on. The deposit of fat in the walls of the gall-bladder, at first along the line of the vessels, but later covering the whole organ, is often the most obvious sign of an infection of the walls.

Gall-stones are found only in the later stages of an infection of the gall-bladder. It is not yet certain exactly where they are formed, whether within the cavity of the gall-bladder or in the mucosa. In the majority of cases they are probably formed within the cavity of the gall-bladder, being due to the clumping of organisms in the bile, and to the protective covering of these organisms by deposits of cholesterol. To impress upon students this truth, I told them long ago that "every gall-stone is a tombstone erected to the memory of the organisms dead within it." But the organisms are sometimes buried alive. Llewellys Barker, of Johns Hopkins, records the case of a patient who, at the age of 8, suffered from typhoid fever, at the age of 43 he was operated upon for gall-stones, from the interior of the stones living active typhoid organisms were recovered.

Much has been written of the 'latency' or the 'innocence' of gall-stones, but with one single exception I believe it to be true to say that gall-stones invariably cause symptoms. Not, it is true, those symptoms of advanced disease which alone were described in the text books of medicine until the present day, but symptoms which are nevertheless sufficiently characteristic.

The one exception to the above rule is concerned with the solitary cholesterol stone which often becomes impacted in the cystic duct. The cause of the formation of this single stone is not yet fully known, but it is I think certain that it is not due, as all other stones are, to infection. Such single stones are found in gall-bladders which show no sign of bacillary invasion, and the bile is constantly sterile, nor can any organisms be found in the centre of the stone. In the later stages, after many severe attacks of pain from the gall-bladder walls may become altered, but such changes are consecutive and not primary. The relationship between this type of stone, and indeed all forms of gall-stone and the cholesterol content of the blood is not referred to here. It is a matter of the greatest interest and importance, but not immediately relevant to the points I wish to raise.

A single cholesterol stone is an ovoid stone rarely larger than a nutmeg. Its surface is finely granulated, on section it presents a number of radiating marks, like the spokes of a wheel. It contains no organisms, and no other constituent than cholesterol. It is sometimes found just beyond the first segment of the valves of Heister. It causes no symptoms until it obstructs the duct, and that is the chief feature which clinically distinguishes it from all other forms of gall-stones. In all of these dyspeptic symptoms are aroused and may be present for months or years before any obstructive symptom develop.

The first indication of the presence of a single cholesterol stone is always a sudden attack of most agonizing pain, beginning in the epigastrium, spreading across the abdomen, and through to the tip of the shoulder-blade. The patient feels as though transfixed by a knife. The agony is terrible, the patient, unable to breathe (the diaphragm being in spasm) feels as though he would burst owing to the great and intolerable distention. Vomiting may bring relief. Relief, however it comes, comes in an instant. This absolutely abrupt onset and absolutely abrupt cessation of agony are quite characteristic of cystic duct obstruction, and are never seen so plainly in any other condition. When the pain persists for a few hours the gall-bladder may be palpable, the area over it remains tender and feels sore for many days afterwards. These typical symptoms, in the absence of any antecedent dyspepsia, enable a diagnosis of a solitary cholesterol stone to be made with a considerable degree of confidence.

All other stones than this are due to infection, and infection, being present before stone formation, may give rise to symptoms which it is slowly becoming within our power to recognize. They are at present, however, suggestive rather than decisive. They are wholly referable to the stomach. Flatulence and fullness after meals, amounting sometimes to so great distress that a woman takes off her corsets or loosens them, early satiety during a meal, a feeling that when a small meal is taken the stomach is overfull, a sudden unaccountable sensation of intolerable nausea, described very often as 'sea sickness' sometimes accompanied by faintness and often by salivation, a feeling of cold associated with slight shuddering, often coming on with great regularity, and 'acidity' and 'water-brash' are often mentioned by the patients.

None of these symptoms is severe, and none striking. It is rather in the association and persistence of them than in their individual character that their importance lies. The complexion of patients is often altered, although they do not realize it. After removal of the infected gall-bladder a patient will often comment upon the improvement in the complexion, and remark that it is 'as it used to be many years ago.' Now and again in such patients a more acute disturbance of health is noted, pain and distress in the upper part of the abdomen are associated with local tenderness, with swelling of the liver, whose edge becomes more easily palpable, and with a slight increase of tenderness. It is as though the whole liver were affected by a slight, but transient, inflammation. Some months, or years, later an attack of hepatic colic occurs, not with the agony associated with the passage of a calculus, but with the rather more subdued but still sufficiently acute pain that probably indicates the passage of bile which is inspissated by thick mucus. In an intelligent patient these several steps may all be traced.

The first cause of these symptoms is uncertain. In recent years inquiry has been made into the association of cholecystitis and hepatitis. E. A. Graham examined portions of the liver removed with the gall-bladder in the operation of cholecystectomy, and found definite changes therein in 87 per cent of the cases. Now and again a fragment of the liver comes away with an adherent gall bladder. In all such pieces we have found changes—advanced or slight—in the liver substance, and have attributed them to an extension to the liver from the gall-bladder. But it appears to be not unlikely that in many cases it is the liver that is first involved in the inflammatory process, and that the gall-bladder is attacked later by invasion of its lymphatics or by direct extension. The inaugural symptoms of cholecystitis may be due to lesions in the appendix, the liver, the gall-bladder, or all of these organs. Our present knowledge does not allow us to decide, but it is the stomach that is always blamed.

Pathology—The changes produced in the gall-bladder by infections which reach it through the bile, the blood, or the lymphatics, produce changes that are slight but easily recognizable by the practised eye. Among the earliest of such changes is a loss of lustre and of colour. The surface is dimmed and whiter, the normal blue colour being lost everywhere except perhaps at the fundus, and the texture of the walls is a little thicker, and suppleness is lost, the elastic layer—as we know by examination of sections—being soon destroyed. A deposit of fat is found beneath the serous surface extending upwards along the vessels first, from the cystic duct. The whole gall bladder is oedematous, and the fundus may show a patch of thickened and reddened opacity which feels almost like a tumour. The cystic gland is enlarged, and sometimes the glands along the common duct also. The pancreas may be enlarged more especially towards the head.

The gall-bladder may be adherent to the stomach, or duodenum or colon. There is, however, no adhesion of the gall bladder that is normal, it is in the form of a mesentery attaching the organ to the duodenum on the inner side and to the colon below. It is probably an extension of the mesogastrium to the right. It is easily recognized. Adhesions which bind the gall-bladder to any neighbouring structure are always evidences of an infection which, wherever originating, has spread at last to the parts around. It is probably true to say that every gall-bladder adherent in this manner has pathological changes so advanced within its walls as to warrant its removal.

When the gall-bladder is opened the bile is thicker in consistency and darker in colour than usual. The mucosa may be œdematous and turgid, and deep red or purple in colour. The villi at first are swollen and sodden, but later are smoothed away. The strawberry appearance is commonly seen. In the later stage erosions, ulcers and diverticula may appear, and little abscesses are sometimes found within the walls. Small shaggy papillomata are not infrequent. I have many times found them so placed that it was possible they had been washed into the cystic duct, and had obstructed it. These papillomata are frequently of a bright yellow colour from the presence of deposited lipid. They often possess an extremely tenuous attachment to the mucosa, and must often become detached. It is reasonable to suppose that under suitable conditions they may become the starting-point of calculi. In still later stages cicatricial tissue is found, and the walls appear thick, hard, and sclerosed. So advanced a change almost invariably depends, however, upon the long-continued irritation of gall-stones.

NOTE ON THE HISTOLOGY OF GALL-BLADDER DISEASE, BY DR O. GRUNER.—A histological study of the walls of the gall bladder which has been made in 100 cases, has shown that the lesions to be found may be grouped according to their relation to the mucosal coat. (1) *Cases showing the chief changes in the mucosa and submucosa*, (2) *Cases in which the chief lesions appear in the subperitoneal tissues*.

When considered in this way, the channel of infection may be readily seen in the microscopic sections as being either by way of the mucosa or the peritoneum—in the former case presumably through the blood stream, and in the latter through the lymphatics. In the cases in which the infiltration is all through the coats, that layer which shows the most intense infiltration is presumably the one in which the infection began, and the peritoneal infiltration in these cases is due to the fact that the organisms are making their way through from the mucosa into the subperitoneal layers by means of lymphatic channels. And furthermore when the whole thickness of the wall is involved in this way, it appears probable that the infection has not been a single event, but has been repeated at least once and very likely many times. This constitutes 'recurrent cholecystitis.'

1 *Mucosal Infections*.—In these cases the early changes noted are œdema of the folds or villi and the appearance of a certain number of inflammatory cells. As the process increases in severity, the œdema spreads through the muscular wall into the subperitoneal layers, and at the same time there is a gradual accumulation of inflammatory cells in the same direction, and fat spaces make their appearance in the subperitoneal tissue.

Should the inflammation subside at this stage, the gall bladder may return to normal, regaining its flexibility and elasticity, though always retaining the tell-tale deposit of fat. But if the process does not come to an end—either because the circulation in the walls is hindered by the presence of stones, or because the invading organisms are of greater virulence—hemorrhages occur from time to time, and a well marked cellular infiltration becomes evident, so that the mucosa becomes very thick. Superficial hemorrhages accompany the formation of ulcers, and the extension of organisms into the walls is accompanied by an interruption in the continuity of the muscle bundles, and a loss of elastic tissue. Once this stage has been reached the viscus can no longer retract and the damaged muscle cannot attempt to expel its contents. Moreover, the soggy nature of the walls makes them incapable of changing their shape, they can only be distended more and more if bile should happen to enter the bladder still further, or contract by reason of cicatrization.

A still later stage, with yet more advanced tissue changes, is reached when the mucosa is converted into a granulation tissue, all the normal structures having been lost. This is sometimes the effect of double infections of the walls, as for instance by streptococci combined with *B. coli*, or by microbial organisms associated with *B. coli*. Sometimes it is the effect of repeated infections by similar organisms each time. This stage may subside by a natural process of organization of the granulation tissue, in which case all structures of the normal wall are absent, and the gall bladder is composed of a mass of fibrous tissue more or less laminated, and enveloped in dense pericholecystic adhesions.

2 *Peritoneal Infections*.—The peritoneum becomes thickened by the intense engorgement of the vessels as well as by œdema, and these changes may involve the subperitoneal tissue also as far as the musculature. In this case the elastic fibres are damaged in an early stage, and a number of changes affecting the mucosa become possible owing to a secondary disturbance of the conditions within the gall bladder lumen. As the acute phase subsides, a fibrosis and permanent œdema of the outer coats becomes evident. Even here a re-infection may occur and lead to the formation of an extensive granulation tissue replacing the original wall, although the mucosa is still relatively unimpaired. The natural result in such a case would also be the formation of a chronic cicatricial contracted gall bladder.

In this brief paper I am considering the question of infection alone. But before we can come to any final conclusions with regard to the formation of gall-stones, other

factors concerned are in need of discussion. Among these the most important is that of the cholesterol content of the blood. Dr McAdam has been working upon this question in connection with some of my cases, and the following note which he has kindly written for me will serve to introduce the subject.

In the course of an investigation into the cholesterol content of the blood in various pathological conditions carried out by Miss C. Shiskin, M.B., and myself, a series of cases of cholelithiasis have been examined before and after operation. Sixty per cent showed a hypercholesterolaemia, while the remainder gave normal values. The latter cases have doubtless shown an excess of blood cholesterol at one time or another, the gall stones present being perhaps the relics of a former hypercholesterolaemia.

A subnormal value was found in a number of cases clinically diagnosed as cholelithiasis not included in the above list, since at operation no calculi were found. Although the presence of infection always tends to reduce the cholesterol of the blood, yet a chronic cholecystitis does not appear to reduce the cholesterol content in conditions of cholelithiasis, and a frankly low pre-operative value should make one suspect some other condition than cholelithiasis except in conditions of acute infection.

The immediate result of operative treatment is a marked fall in the cholesterol of the blood. This is most marked in cases of drainage of the biliary passages. The effects of the anaesthesia doubtless also play a part, while we have observed in all the conditions investigated, other than cholelithiasis, that operative procedures seem invariably to lead to an immediate loss of cholesterol in the blood.

But it is the cholesterol value of the blood some months after operation that appears to be of practical importance. Rothschild and Rosenthal have distinguished two types of hypercholesterolaemia in cases of cholelithiasis.

1 *Obstructive hypercholesterolaemia*, which is temporary, the cholesterol content of the blood returning to normal with the removal of the obstruction.

2 *Dietetic hypercholesterolaemia*, in which the excess of cholesterol is more or less continuously present. This condition may be intensified by the additional presence in the bile passages of an obstruction to the completion of the metabolic cycle of the cholesterol. When there is this diathesis, the hypercholesterolaemia persists, even after the removal of the obstruction. Most of the cases, so far examined by us, belong to Group 1, a few, however, had a markedly high cholesterol content several months after cholecystectomy. These findings suggest that, in cases which show a distinct hypercholesterolaemia before operation, provision should be made for drainage of the bile in order to deplete the body of the retained lipoids. If a later examination should reveal a persistently high cholesterol content, then further accumulation of cholesterol may be controlled by dietetic measures.

Recent work has shown conclusively that there is no synthesis of cholesterol in the body, and that any addition to the total cholesterol content of the blood and tissues is derived from that present in the food. Free cholesterol is converted into cholesterol esters in the intestinal canal, from which they are absorbed and are distributed by the blood stream to the body cells. As the result of metabolic activity in the cells, cholesterol is again liberated, carried by the blood stream to the liver, and excreted into the bile, to be again re-esterized and re-absorbed from the intestinal tract. Thus an interference with this constant cholesterol metabolic cycle through drainage of the bile, in cases of dietetic hypercholesterolaemia, may be of considerable surgical importance.

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A COMPARISON OF CHOLECYSTOSTOMY AND CHOLECYSTECTOMY.

By JAMES SHERREN, CBE, LONDON

THIS problem can be stated very briefly indeed, for the change in opinion that has steadily taken place in the last few years puts it now almost beyond discussion. It was formerly the custom to remove the gall-bladder only if the surgeon considered it so seriously affected as to interfere with its function, and this decision may have been arrived at on external examination only. In this way many cases of disease remained unrelieved and many stones were overlooked. It is impossible to prove the absence of stones unless the gall-bladder has been opened. Inflammatory affections and simple growths are even more easily missed unless this precaution is taken.

Ten years ago, in 100 consecutive operations for gall-stones, I considered cholecystectomy necessary in 29. In my last 100 I carried it out primarily in 94, the other 6 were acute cases in which it was unwise for various reasons to make the attempt, and in two of these I removed it at a second operation three weeks later. This change in practice has been forced upon me by experience.

I wish to state at once that in my opinion the results, both immediate with regard to death rate and convalescence, and remote in the freedom from complications and absence of recurrence of symptoms, are infinitely superior if the gall-bladder is removed as a routine in the treatment of its diseases. This is not the doctrine to preach to those whose operative interference with the biliary passages is and will be occasional, to these the simpler operation of cholecystostomy is the safer. The skilled surgeon however must now justify his choice of cholecystostomy, not that of cholecystectomy.

The disease of the gall-bladder is what we should treat, it is not enough to remove the products of that disease whether gall-stones or infected bile. I can state emphatically that drainage does not cure chronic cholecystitis.

Cholecystectomy should be the treatment in all surgical diseases of the gall-bladder as a primary procedure where possible, but in two stages in those cases where the risk to life or the common duct is great. I refer to such cases as acute or chronic cholecystitis, where there is an enormously distended, thickened, and adherent gall-bladder containing pus, acute cholecystitis or stone in the common bile duct with jaundice. We all try to avoid two operations on one patient whenever possible, but I always remind myself that it is better to have a live patient after a two-stage operation than sign the death certificate for the result of an 'ideal' and that cholecystectomy as a second procedure is infinitely preferable to a plastic operation on the common duct.

The experience which led me to consider cholecystectomy necessary was obtained from the examination of gall-bladders removed at operation and comparative results after a period of years.

It is as unwise to diagnose the condition of the gall-bladder by external examination as it is to say in the absence of definite x-ray evidence, that the symptoms from which a patient is suffering are due to gall stones rather than a chronic cholecystitis or other cause of biliary colic. I have made it a rule never to tell patients they are suffering from gall stones, but from inflammation or disease of the gall-bladder. We should endeavour to diagnose the disease, not its results.

In many cases in which external examination of the gall-bladder and examination of the lymphatic glands in the neighbourhood have shown nothing abnormal, cholecystostomy has revealed not only small calculi, but such conditions as that in which crystals

of cholesterol or tiny calculi are embedded in the mucous membrane, chronic cholecystitis of the 'strawberry' type, or the thickening met with in the fundus of the gall-bladder called adenoma. In these a mass of adenomatous material occupies the fundus, occasionally becoming cystic. Seen from its mucous aspect it often shows a curious umbilicated appearance. It is occasionally associated with chronic cholecystitis of the 'strawberry' type. Its presence may give rise not only to the usual secondary dyspepsia, but to typical attacks of biliary colic and I have removed the gall-bladder for this condition in seven patients who had no calculi, with cure of the symptoms. I have met with it in several associated with gall-stones, and in two, although the naked-eye appearance was identical with those I have referred to, microscopic examination proved them to be columnar celled carcinoma. Both have remained free from recurrence, one operated upon nine, and the other two and a half, years ago. Polypoid cholecystitis may also be overlooked if cholecystostomy is carried out. Drainage in cases such as these must fail to cure. All need treatment by removal of the gall bladder.

It is, however, on the ultimate success of our procedures that we must base our practice. What are the results of the two operations? I do not intend to do more than mention those complicated by stone in the common duct. My figures are small compared with those that could be obtained from many clinics, but all have been carefully followed up.

Taking first the cases of cholecystectomy carried out in the treatment of stones confined to the gall-bladder and operated upon over three years ago, these number 184, with 6 deaths, 3 of which were from lung complications, and include 33 operated on in the acute stage. There has been no recurrence of symptoms in any of these cases. I do not believe that stones, apart from the rare pigment variety in certain diseases of the spleen, form in the common duct after cholecystectomy if that duct is clear at the time of operation.

Taking now the cholecystostomies for stone done during the same period, 152 in number, including 46 acute cases, there were 4 deaths. Of the acute cases 8 have had definite recurrence of symptoms. Of the 106 non-acute no less than 21 have had similar relapses. In 75 per cent of the 29 patients who relapsed symptoms returned within two years, in the remainder it was from four to nine, and it was while preparing this paper that I have had to operate on recurrences seven and nine years after drainage. I have re-operated upon 18 of these patients. In 3, there was chronic cholecystitis only, in the remainder stones had re-formed. In none were stones present in the common bile duct, nor were stones found here at the second operation. In addition to these recurrences, 5 patients died within four years of carcinoma, certainly or probably of the gall-bladder. In one I removed the gall-bladder a year later, but the patient died of recurrence in a few months, in another the patient returned in twelve months with an obvious malignant gall-bladder tumour and ascites, another in six months, and 2 died, one two and one three years after operation, of cancer of the liver. In none was the gall bladder left because of the condition of the patient, but because I did not consider it was grossly diseased, justifying removal. This gives a total of 34 out of 148 survivors in whom the result was unsuccessful after a period of years.

I cannot believe that these figures are exceptional. It is not a difficult task to clear the gall-bladder of stones, and I believe if all cases were followed up as these have been, similar results would be obtained. I have communicated with the patient or his doctor at least once a year and have not lost trace of a single case.

During the period under review, out of 448 primary operations for gall stones, in 83 I removed stones from the common duct and in 29 carcinoma of the gall-bladder was found to be so extensive that operative treatment was impossible.

During the same period I also carried out cholecystectomy for disease not associated with stones, in 30 chronic cases without a death and complete and permanent relief of symptoms, and in 9 acute cases with 4 deaths, all cases of gangrene of the gall-bladder.

I have excluded from the discussion those cases in which I removed stones from the common bile-duct, as it is notoriously difficult to be certain that the ducts are clear. It

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is my practice, whenever possible, to remove the gall-bladder and, if drainage is necessary, to drain the duct itself. My re-operation rate was nearly three times as great when the gall bladder was not removed.

I have not attempted to go into theories with regard to the function of the gall-bladder and changes inimical to the well being of the patient that may result from its removal. I have given you facts drawn from my own clinical experience that to my mind leave no doubt or room for discussion that the correct treatment of gall-bladder diseases, including in that term gall-stones, is cholecystectomy, although in certain cases drainage may be necessary as a temporary measure. Cholecystectomy is safer for the patient, the risk of recurrence is negligible, and the loss of the gall-bladder interferes in no way with his well-being.

VISITS TO SURGICAL CLINICS AT HOME AND ABROAD

SOME DUTCH SURGICAL CLINICS

THE CLINIC OF PROFESSOR NOORDENBOS, AMSTERDAM

ONE of the Surgical Clinics of the University of Amsterdam is situated in the Municipal Hospital at the Binnen Gasthuis and includes 110 beds, together with out-patient departments

Professor Noordenbos directs this service with the help of four qualified assistants. The large and well-lit operating theatre is specially adapted for demonstration purposes.



FIG. 110.—Professor Noordenbos in his operating theatre.

by having a number of seats for students arranged in semicircular tiers (Fig. 110). In this place he demonstrated his method of treating fractures of the neck of the femur, showing one man of seventy on whom it had recently been performed (Figs. 111, 112). A longitudinal incision is made in the outer aspect of the great trochanter, the leg is held with strong abduction, traction and internal rotation, and a large twist drill is entered on the shaft of the femur 2 cm. below the most prominent point of the great trochanter,



FIG 111 —Fracture of the neck of the femur (before operation)



FIG 112 —The same case as Fig 111 (after operation)

directed to the anterior superior spine of the opposite side. Into the hole thus made a portion of the fibula $8\frac{1}{2}$ cm long is driven, together with its periosteum. The limb is put up in an abduction plaster for three months, and the patient is not allowed to walk for five months. During the later portion of this period—that is, after the removal of the plaster—the patient takes exercise on a little trolley frame which runs on wheels on the bars of a special frame bedstead, sitting on the trolley with both feet on the lower bar of the bedstead, pulling himself down by a cord and pushing himself back by straightening the leg as is done by a rower on a sliding seat.

Professor Noordenbos has done this operation fifty-four times without failure. He prefers to do it in recent intracapsular fractures, but has performed it with equal success in old cases with pseudarthrosis.

Among other interesting cases demonstrated in the theatre were the following —

Resection of the Stomach for Ulcer associated with Internal Hernia — A man of fifty-two presented himself with abdominal pain and distention, associated with severe tetany. He had a history of thirty years' dyspepsia, during twenty-five of which he had obtained some measure of relief by washing out his own stomach. The abdomen was opened by a transverse incision on the right side between the sternum and navel. A complicated condition of retrogastric hernia greatly obscured the anatomy of the parts and hid the stomach from view. A number of coils of small intestine had prolapsed through an opening in the transverse mesocolon and then pushed forward the thin lesser omentum, hanging downwards over the anterior surface of the stomach. The hernia having been reduced, a large indurated ulcer was discovered at that point on the lesser curvature which had been crossed by the small intestine, and there was also a tight cicatricial stenosis of the first part of the duodenum. A partial gastrectomy was performed by the Balfour Polya method.

The patient, whom we saw a week after this operation, was making an excellent recovery.

Substitution of the Œsophagus by the Jejunum — A girl of ten had suffered occlusion of the Œsophagus as the result of swallowing caustic potash. Attempt at restoring the canal by bougies having failed, a gastrostomy was performed, and, at a later stage, a loop of 30 cm of jejunum, having been isolated from the rest of the gut, was attached to the stomach and brought up to the skin in front of the sternum. It was at this stage that we saw the patient, who was awaiting a final plastic operation for the junction of the jejunal fistula to the pharynx by means of a tube of skin, to be fashioned from the chest wall.

Two Cases of Laryngectomy — In both cases the operation had been done for epithelioma and had been performed under local anaesthesia. The trachea had been brought out through a separate incision and attached to the skin. One of these cases of laryngectomy was a young man of thirty-three, who was so pleased with his relief from impending asphyxia, and so delighted to be shown to a party of English surgeons, that he wrote on a piece of paper "This is our Lloyd George, he does our reconstruction."

We witnessed the following three operations —

1 Bone-graft for Spinal Caries — (*Operation 9.15 to 9.45 a.m., commencing twenty-five minutes after the anaesthetic*) The patient was a boy of seven with caries of the fourth, fifth, and sixth dorsal vertebrae. The disease was in an early stage without deformity. The lesion was beautifully shown by means of a radiogram taken with the assistance of a Bucky-Porter diaphragm. Before the operation the patient is accustomed to lying prone on his face, in which position the operation is done, and this position is maintained throughout convalescence, no other splint or immobilization appliance being used. The patient is nursed on a special narrow mattress the width of the trunk, designed to allow free movement of the arms.

The operation was performed under local anaesthesia by a solution of $\frac{1}{2}$ per cent novocain with adrenalin (novocain 0.5, K_2SO_4 0.4, NaCl 0.7, water to 100, adrenalin 1.2 drops to 100 c.c.) The anaesthetic was injected both superficially and deeply round

the affected area of the spine and over the tibia from which the graft was to be taken. The process of injection, which occupied about a quarter of an hour, made the child cry bitterly. A curved incision was made on one side of the spinous processes from the third to the seventh dorsal vertebræ. The muscles were separated from the left side of the spinous processes, which were then sawn off from the laminae by means of a small Ferguson's saw aided by a chisel and forceps. The spines thus separated were left in attachment to the muscles of the right side, and were then pushed towards the right whilst the posterior surfaces of the laminae were further sawed. The wound was packed whilst

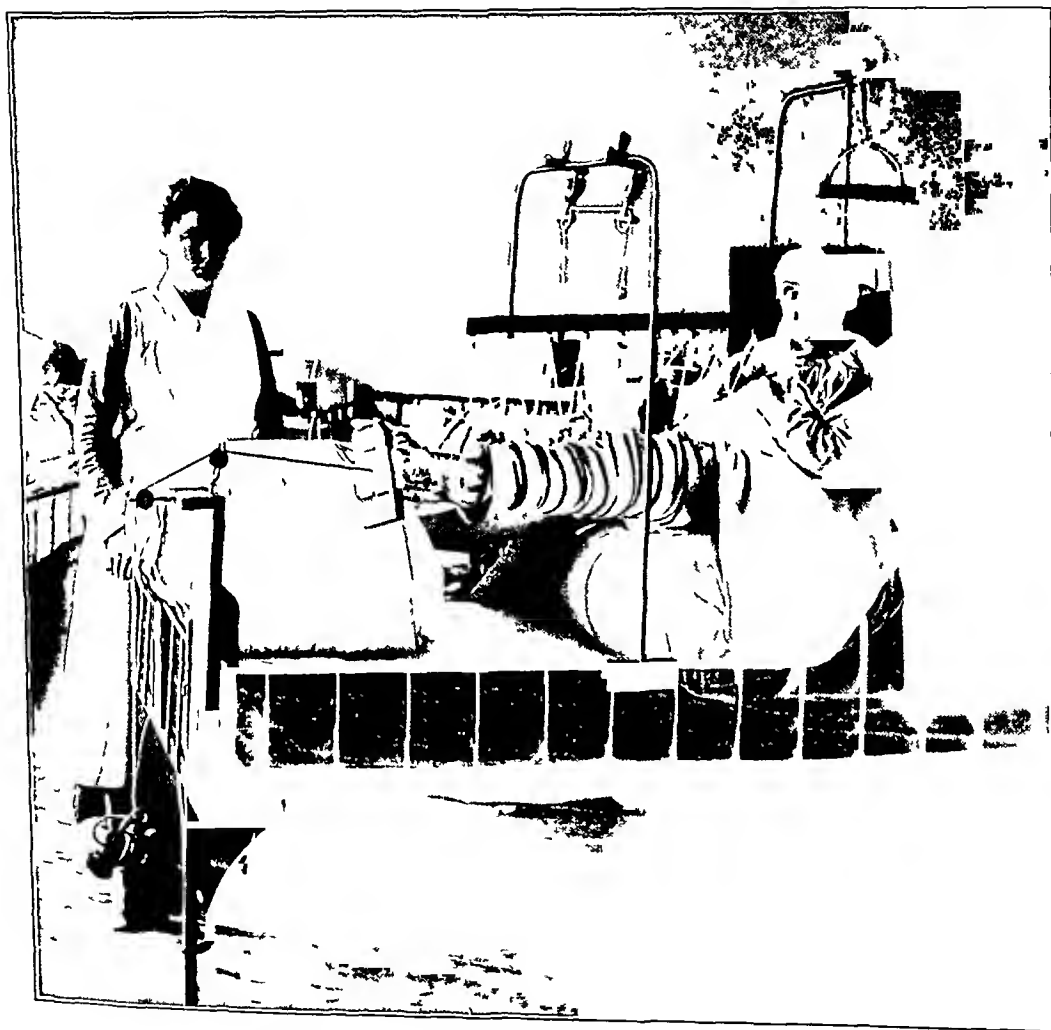


FIG. 113.—Illustrating the suspension method adopted for fracture cases in the Noordenbos clinic.

the graft was being cut. The right leg was fixed at the knee and the tibia exposed by a curved incision. Two cuts were made obliquely into the bone for a depth of about three-quarters of an inch, and at a distance of about five inches from one another, by means of a large bow saw. A number of transverse drill-holes were made from side to side through the whole thickness of the bone between the two saw-cuts, and a piece of tibia including the whole interosseous surface five inches long and three-quarters of an inch wide, was removed by a chisel. The actual cutting of the graft occupied six minutes. The graft was placed with its marrow surface next to the laminae, the spines were brought over it,

and there fixed by deep catgut sutures. The aponeurosis and the skin were separately sutured. The child's colour at the end of the operation was excellent.

2 Calculus in the Right Ureter—(*Operation 10 16 to 10 55 a.m.*) A woman, age 45, had pain in the right loin, shooting down towards the bladder. The radiogram showed an oval calculus in the position of the right ureter, and this had been confirmed by the passage of a lead ureteral bougie, the point of which was arrested where the shadow of the stone was shown. The anæsthetic was by open ether after preliminary morphine-atropine-scopolamine. An oblique incision 8 inches long was made parallel with Poupart's ligament. The operative field was surrounded by dark blue cloth and a forehead light used to illuminate the deep wound. The ureter was fully exposed and lifted up from the point where it crossed the pelvis to the base of the bladder. No stone was discovered and it was therefore concluded that it must have been dislodged after the passage of the lead bougie. The wound was closed in layers by interrupted silk sutures.

3 Prostatectomy under Sacral Anæsthesia—(*Anæsthetic 11 10 to 11 40 Operation 11 40 to 11 55*) The patient was a man, age 55, who for some years had had myelitis with incontinence, but for the last twelve months had suffered from retention of urine associated with prostatic enlargement.

Patient lay face downwards on the operation table with his legs flexed at the thigh. A point was taken on each side where the sacrum and coccyx join and a needle 13 cm. long pushed along the anterior surface of the sacrum, injecting 1 per cent novocain all the time. Fifty cc. of novocain solution were injected into each side so as thoroughly to infiltrate the nerves emerging from the anterior sacral foramina. The patient was then turned on his back, and the median subumbilical region was surrounded by an anæsthetic injection, this being given first under the skin and then into the rectus sheath. A catheter had been tied into the urethra, and through this the bladder was distended with air. The operator stood at the patient's right side and enucleated the prostate with the left hand the right being engaged in the rectum. After removal of the gland, which was a symmetrical fibro adenoma about one inch and a half in diameter the bladder was drained by a double rubber tube, a gauze pack being left in the space of Retzius. The rest of the wound was closed in three layers, the deep layers and skin by silk, and the fat layer by catgut. The anæsthesia in this case appeared to be perfect.

The silk which is so freely buried, even in the tissues of wounds of doubtful sterility is prepared as follows: ether one day, 80 per cent alcohol one day, boil half an hour in 1-1000 sublimate, store in 1-1000 sublimate in alcohol.

A short visit was afterwards paid to the wards and a great number and variety of cases were seen. There were a large number of fracture cases most of them being treated by a method of suspension and traction, which appeared to be very effective. The limbs were suspended to a metal frame clamped on to the bed. The upper limb was slung in a position of abduction of the humerus with right-angle flexion of the elbow, the limb hanging with the forearm vertical traction being made on the humerus by transfixion of the olecranon. The lower limb was slung to an anterior wooden bar by means of a cord which was attached to a number of points along the leg by a bandage and brass rings the whole apparatus being strongly suggestive of a piece of ship's rigging (*Fig. 113*).

DR J. SCHOEMAKER AT THE HAGUE

'Operative surgery is not a sport it is an art and just as a violin player plays his sonata with his heart and soul so the surgeon must perform his operation. This means that the artist does his work not in a hurry not slowly but in tempo. It also means that within these laws he is at liberty to do his work in his own way striving for perfection and beauty, so that the finished product may be a work of art. But the surgeon is not a solo player, he is the first violin of a quartette the other members of which are his

assistant, his anesthetist, and his operation nurse' (*Surgery Gynecology, and Obstetrics*, 1921 Dec, 591 from which journal the accompanying drawings have been copied)

When Dr Schoemaker uttered these words before the Clinical Congress of American Surgeons, they were probably regarded as a somewhat high-flown hyperbole but anyone who watches him at work will be forced to admit that they represent most accurately the actual manner and method of his work

We saw him do eight major abdominal operations on two successive mornings, and he had selected the particular cases in order specially to illustrate his own methods of performing colectomy and gastrectomy. Cases 1, 3 and 6 were instances of colectomy and it will save repetition if the technique of this operation is described once.

Case 1—(Operation 10:15 to 11:0 a.m.) The symptoms were those of constipation the patient being a middle aged woman

Case 3—(Operation 12:5 to 12:45 p.m.) The patient was a woman, age 28 who had suffered from pain and constipation for eight years

Case 6—(Operation 10:15 to 11:0 a.m.) A woman, age 63 who had severe bleeding from the rectum with pain on the right side of the abdomen

In all three cases there was marked dilatation and mobility of the cecum and ascending colon. In the last case there was a large inflamed appendix

The abdomen was opened by an incision through the right semilunar line and the wound held open by a large self-retaining retractor. The ileum was held up in its terminal part, and a hole made in its mesentery. The outer layers of the bowel were divided about three inches from the cecum and a cuff consisting of these layers was separated from the mucous

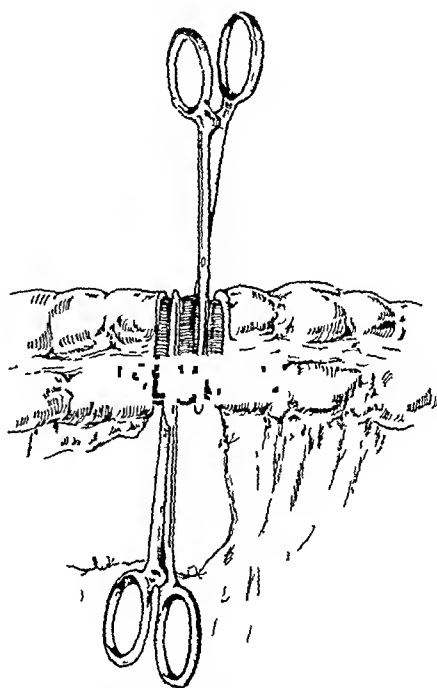


FIG. 114.—Retraction of the colon. Location of the clamp.

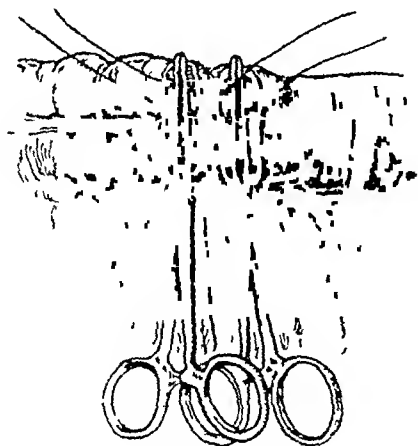


FIG. 111.—Holding the sutures in the incision.

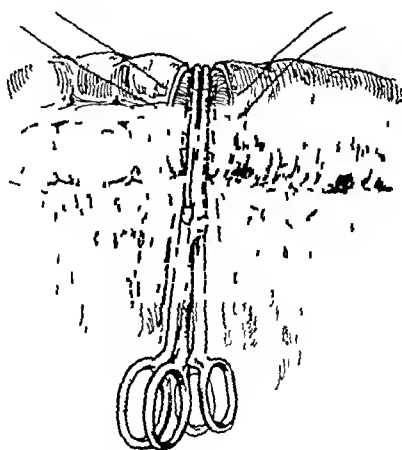


FIG. 112.—Closing the incision by a continuous suture.

membrane for a distance of about half an inch. The mucous liver thus isolated was clamped by two small forceps shaped like a Kocher's forceps but having deep longitudinal grooves on the inner surface of their blades. The bowel was divided between the

forceps The ascending and proximal part of the transverse colon were separated from the outer leaf of peritoneum and from the great omentum by means of a few touches with the scissors, the main vessels going to the ascending colon and hepatic flexure were clamped and tied in four places The transverse colon was divided in the same way as the ileum, first by cutting through the outer walls of the bowel and pushing them aside, then clamping the mucous membrane between two forceps (*Fig 114*), and cutting between these The separated portion of the bowel was removed, and the ileum brought into contact with the distal portion of the transverse colon, the two portions of bowel being held in apposition by the attached clamps

It was very noteworthy that after division of the outer wall of the gut, the lumen of the ileum and that of the colon were made of the same size, the former being a little stretched whilst the latter was allowed to contract An end-to-end anastomosis was made as follows (*Figs 115, 116*) The assistant held the ends of the gut up in a vertical position by means of the clamps, six interrupted silk stitches united the serous coats of the bowel deep to the forceps and just beyond the edge of the cuff, while a second row of suture united the edges of the cuff, taking also a bite into the mucous layer The ends of the bowel were then turned towards one another, and the superficial layers of the cuff were sewn together over the forceps Finally, six more interrupted silk stitches united the serous coat in front of the forceps, the last stitch being tied after the forceps had been withdrawn, so that at no time was the lumen of the gut openly exposed The edge of the mesentery of the ileum was united by a few stitches to the wall of the transverse colon The retractor was removed and the abdominal wall closed in three layers of interrupted sutures, the peritoneum and the skin by catgut, and the muscle by iodized silk

Partial Gastrectomy—(*Operation in one case 11 15 to 12 0 noon, in the other 11 10 to 12 0 noon*) The second and seventh cases were both young women suffering from an indefinite type of dyspepsia The former had been diagnosed as a case of gastric ulcer, but the radiogram gave no definite evidence of this lesion The latter case presented symptoms of dyspepsia characterized by hunger pangs In both cases an inflamed and adherent appendix was found and removed after the stomach had been partially excised In each case the stomach was rather large and prolapsed, the pyloric segment being red, vascular and irritable

The abdomen was opened by a median incision from the sternum to the umbilicus, and the wound was held open by a self-retaining retractor The duodenum was lifted up and the attachment of the omentum clamped and cut in sections Two small clamps were placed on the first part of the duodenum, which was then divided by the knife The distal end was covered by gauze, whilst the proximal end was protected by a little metal shield which fitted on to the clamp The remaining portion of the small omentum was clamped and cut in sections the coronary artery being divided in the last A large pair of special clamps (*Fig 117*) constructed in two portions was then applied to the body of the stomach, the blades of these clamps, which are about five inches long, are curved in about the same shape as the normal lesser curvature of the stomach When in position the blades extend from a point on the lesser curvature of the stomach opposite to the coronary artery, at the junction of the middle and upper thirds of the stomach to a point about an inch and a half from the greater curvature of the stomach and two inches from the pylorus The portion of the stomach between the right end of the stomach clamp and the greater curvature was seized by a pair of small forceps like those for colectomy The stomach was then cut through by a knife applied close to the clamps large and small Thus freed the pylorus and lesser curvature of the stomach which were removed The large stomach clamp consists of two portions After cutting away the pylorus the distal portion of the clamp is unscrewed and slipped out, thus leaving a compressed edge of stomach wall rather more than one-eighth of an inch in extent projecting from the remaining portion of the clamp (*Fig 118*) This projecting edge was sewn over by a continuous catgut stitch, and the remaining portion of the clamp was then taken away and a second continuous Lambert stitch completed

the closure of this portion of the gastric wound. The stomach had now been reduced to a more or less tubular structure, the end of which was closed by one small colon clamp. This was brought into apposition with the duodenum, and after two more clamps had been applied proximal to the gastric and distal to the duodenal forceps, an end-to-end junction was effected (*Fig 119*). The deep surfaces of the viscera were joined by interrupted silk sutures. The terminal clamps were taken off and the whole thickness of the stomach and gut united by a series of interrupted stitches, whilst the anterior layer was completed after the remaining clamps had been removed.

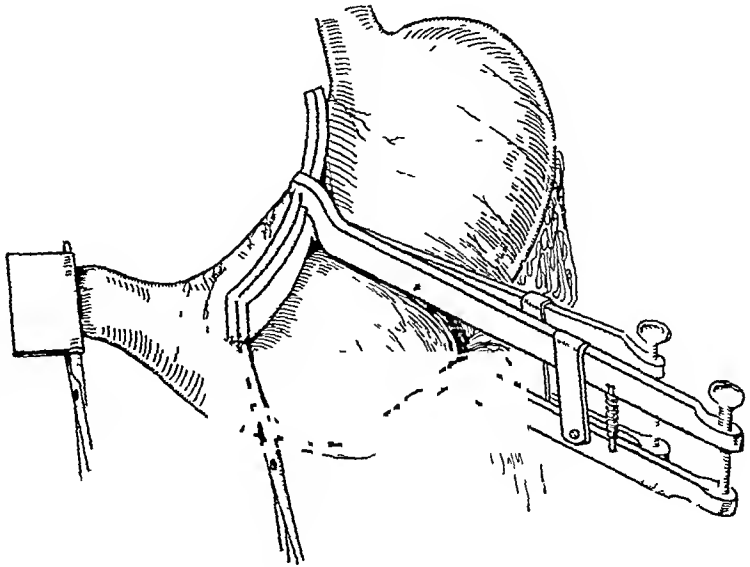


FIG 117—Resection of the stomach. Schoemaker's clamp

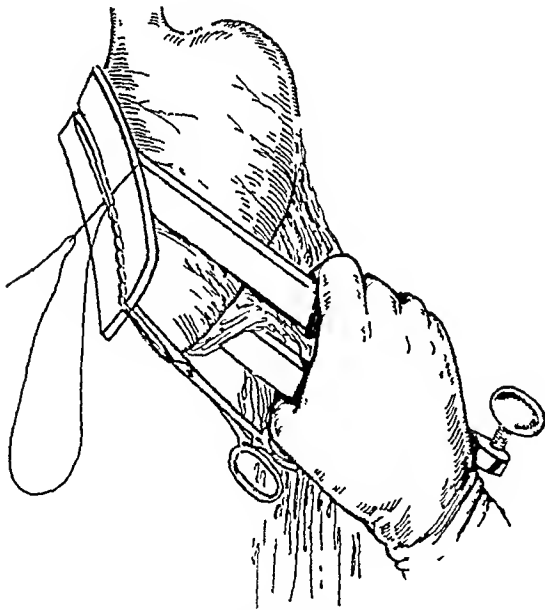


FIG 118—Anterior plate of clamp removed, showing narrow strip of gastro-sutured by a continuous circumferential suture

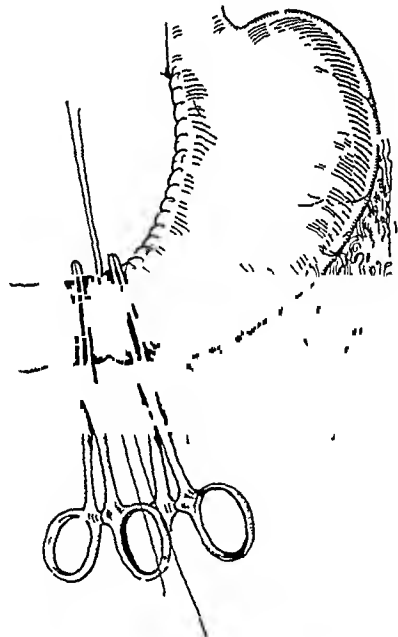


FIG 119—Union between the stomach and duodenum

Cholecystectomy—(Operation 12.50 to 1.15 p.m.) The patient was a man, age 34, who had had symptoms for the last two years. The radiogram showed stones in the gall bladder. The abdomen was opened by splitting the right rectus muscle, the wound was held open by a self-retaining retractor. A large inflamed gall-bladder was found, with a well-marked membrane between it and the hepatic flexure of the colon. After division of the latter the gall-bladder was separated from the liver by blunt dissection.

aided by a few touches with the scissors. The cystic duct was isolated and clamped. An opening was now made into the duct proximal to the clamp, and through this opening a stone was removed from the common duct, a metal sound like a uterine dilator was first passed down the common duct in order to dilate the duct and facilitate removal of the stone from its lower end. The gall bladder was removed and the opening in the duct sutured by interrupted catgut. These stitches were left long, and threaded through a large rubber drainage tube which was inserted down to the duct. The wound was closed in three layers.

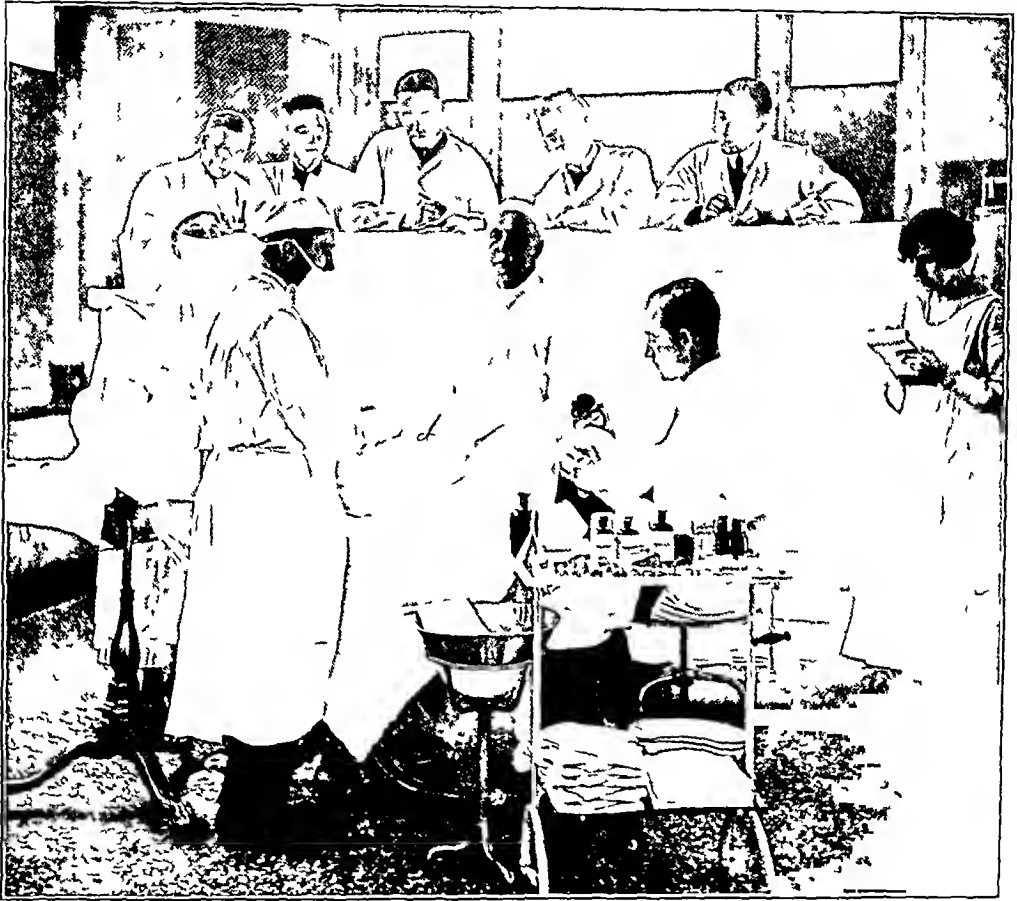


FIG. 120.—The operating theatre in Dr. Schoemaker's clinic illustrating the position of the patient and the assistant.

Gastro-enterostomy for Stenosis of the Pylorus—(Operation 9.40 to 10.10 a.m.) The patient was a man age 66, who had been suffering for some time with dyspepsia and vomiting. A provisional diagnosis of cancer of the pylorus had been made because of the absence of HCl, the presence of lactic acid and the abrupt limitation of the x-ray shadow. The abdomen was opened through a median incision and the pylorus found to be densely indurated and adherent, but the appearances were not those of a carcinoma. A retrocolic gastro-enterostomy of the usual type was performed. The stomach was not clamped, but the jejunum was isolated by two separate clamps. The outer row of suture was continuous silk and the inner continuous catgut. In placing the latter each edge of mucous membrane was pierced twice by the needle so as to leave a loop on the deep surface and produce an infolding of the edge.

Nephrectomy—(*Operation 12 15 to 12 40 p m*) The patient was a stout elderly woman who had had lumbar pains for several years and in whom a radiogram showed a large branching calculus. The kidney was removed through a long oblique incision, and on cutting it open afterwards several smaller stones were found, with suppuration and atrophy which had almost destroyed the cortex.

In all these abdominal operations Dr Schoemaker maintained the same general arrangement of the table and his assistants (*Fig 120*). The patient's legs were always lowered a perineal prop supporting the weight, the instrument table presided over by the operation nurse was placed across the patient's feet. The operating quartette thus faced the patient the whole time—the anaesthetist and instrument nurse at the two ends, the surgeon and his assistant on the right and left side. The three persons engaged in the operation wore rubber gloves over which cotton gloves were fastened by a rubber band at the wrist. All changed the cotton gloves after concluding any septic stage of an operation such as the opening of the intestine or stomach. The skin was prepared by a strong tincture of iodine, and during the operation there was a notable absence of any packing in of gauze or swabs, complete reliance being made on the efficiency of the clamps. Hemostasis was remarkably efficient, and a spurting vessel was hardly once seen in the course of the eight major abdominal operations.

PROFESSOR LAMERIS, UTRECHT

Professor Lameris has been head of the University Surgical Clinic at Utrecht for over twenty-five years. He has one pavilion in the up-to-date Polyclinic of that town with 135 in-patient beds and a large out-patient department. He has ten assistants for this work.

The operating theatre was large and well lighted, the whole north wall being glass, there is also an arrangement of an arc light which can be reflected by means of mirrors on to the patient. Dr Lameris uses no antiseptics other than water, soap, and alcohol, gloves are not worn except in the performance of septic operations.

Radical Cure for Inguinal Hernia—(*Operation 11 7 to 11 18 a m*) A young man with a left inguinal hernia. A two-inch incision was made parallel to and above the inner end of Poupart's ligament, the external oblique was divided, the cord was lifted up, the neck of the sac isolated and divided, and the distal end ligatured without removal. The proximal portion of the neck of the sac was twisted a number of times, clamped, transfixed, and ligatured with silk. The stump was dropped back into the abdomen. External oblique and skin were united by two rows of interrupted silk sutures. The patient is kept in bed for about a week. Dr Lameris said that he had operated upon 1200 cases by this method with satisfactory results. He does not, however, operate upon direct hernias, but treats them by a truss.

Gastro-enterostomy for Pyloric Cancer—(*Operation 11 22 to 11 40 a m*) A man, age 70 with a history of pain and hæmitemesis. The abdomen was opened by a five-inch median incision, and an indurated mass found in the pylorus and lesser curvature of the stomach. A retrocolic gastro-enterostomy was performed with the assistance of a curious instrument called a gastrophore invented by Nyrath, of Heidelberg. It is a kind of clamp, the deep blade of which is a convex ovoid and the superficial blade of which is a ring. By means of this clamp the stomach is pressed up against the transverse mesocolon and a bloodless field secured for the attachment of the jejunum. The anastomosis was made by means of three rows of sutures, each being continuous silk. The abdominal wall was closed in three layers, the deep layer by continuous silk, the aponeurosis and skin by interrupted silk.

Recurrent Appendicitis—(*Operation 11 45 to 12 5 p m*) The abdomen was opened by a five-inch incision through the right semilunar line. The mesentery of the appendix

having been clamped and ligatured, the stump was turned in by three separate sutures. The abdomen was closed by three layers of interrupted silk stitches.

Nephrectomy for Pyonephrosis—(*Operation 12 10 to 12 40 p m*) The patient was a woman, age 26, who for the last month had had a hectic temperature and pus in the urine. On two occasions a catheter had been passed into the right ureter for 16 cm. and the kidney irrigated through this, with a temporary relief of her symptoms.

A ten-inch incision was made through the right lumbar region, the patient lying on an air cushion. The kidney, which was very adherent, was isolated by a blunt dissection and removed together with about six inches of thickened ureter. The wound was irrigated by a solution of hydrogen peroxide, which was drawn off by means of a nozzle attached to a water pump. The wound was closed with buried silk sutures and drained by a large tube.

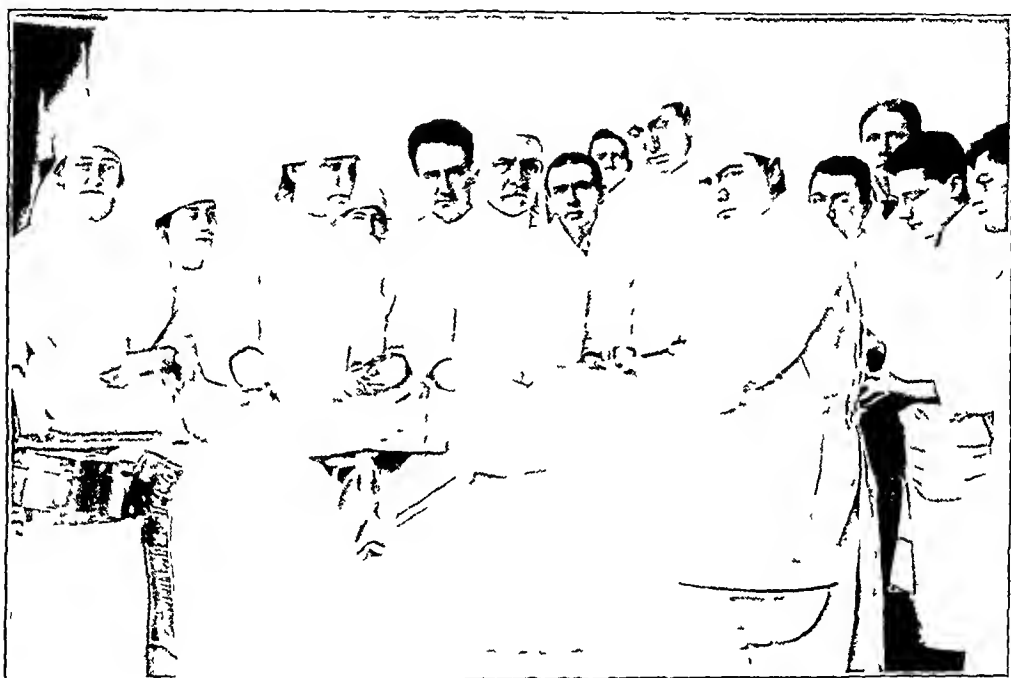


FIG. 121.—Professor Jamieson in the operating theatre.

A Resection of Simple Goitre—(*Operation 9 0 to 9 30 a m*) The patient was a woman, age 35, with symptoms of dyspnoea and a large symmetrical goitre about the size of two fists. The anaesthesia was by local injection of $\frac{1}{2}$ per cent novocain. A transverse collar incision was made. The inferior thyroid artery was exposed and ligatured on each side in the space between the thyroid gland on the medial side and the sternomastoid and carotid sheath on the lateral side, by the method of de Quervain. The hyoid muscles were cut across and turned upwards. The superior thyroid vessels were dealt with by ligating the anterior and posterior branches close to the gland. The superior parathyroid bodies were clearly seen on both sides. The isthmus of the gland was separated from the trachea and cut through. The greater portion of both lobes was then resected leaving a piece of gland tissue on each side about the size of the top joint of the thumb. The cut edges of the remaining portion were then brought together by catgut. The hyoid muscles and skin were united by separate silk sutures, a drainage tube being inserted.

Resection of Cancer of the Colon—(*Operation 9 37 to 11 5 a m*) The patient was a young man, suffering from abdominal pain and constipation and the passage of blood and mucus.

A hard elastic tumour could be felt in the left lumbar region. The abdomen was opened by an incision in the left semilunar line, the tumour being found apparently in the descending colon. As it felt like a polypoid growth and the wall of the bowel was healthy, an incision was made into the bowel, after lifting it up and packing round with gauze. The bowel was opened longitudinally by a three-inch incision, the tube connected with the suction pump being brought into frequent requisition in order to remove septic material. A fungating tumour about two inches by two and a half inches was found to be growing from higher up in the colon, forming the apex of an intussusception. The incision into the gut was temporarily closed by several forceps, and the invagination reduced. After this had been done the tumour was found to be situated in the distal portion of the transverse colon. The splenic flexure together with the contiguous parts of the transverse and descending colon were mobilized by dividing the peritoneum and blood vessels along their medial aspect.



FIG. 122.—In the words

A side to side anastomosis was made between the transverse colon and the descending colon using the opening already made into the latter for this purpose. The anastomosis was performed by means of three rows of continuous silk suture. After this had been done, the portion of bowel which lay beyond the anastomosis containing the tumour was removed by two transverse sections, each of which was closed by three layers of silk. Before closing the abdomen it was observed that a secondary growth existed in the liver. The wound was closed without drainage.

Thoracoplasty for Empyema—(Operation 11.15 to 11.55 a.m.) A young man who had suffered from empyema five months ago. This had been treated by aspiration only, and a sinus remained in the posterior axillary line in the ninth intercostal space. A large flap of soft tissue was turned upward so as to expose the whole left lateral aspect of the chest. Five or six inches were then removed from six consecutive ribs, that is, from the sixth to the eleventh inclusive.

The sinus was excised with the actual cautery. A cavity of six inches by three inches was exposed, and a part of its external wall removed.

The wound was flushed with water, and from the bubbling which occurred it appeared that there must be a communication between the empyema cavity and the lung. The wound was closed by interrupted silk sutures, a large drainage tube being left in position.

Operation for Osteomyelitis—(*Operation 12.5 to 12.20 p.m.*) The patient was a boy age 18, who for ten days had had pain and swelling in the left thigh, chiefly in its lower third. An incision four inches long was made into the external aspect of the thigh, and a cavity containing pus was discovered at the back of the lower end of the femur. The medulla of the femur was opened by a large electric burr, and pus exuded from this opening, which was then enlarged until it presented a hole three inches long and three-quarters of an inch wide. Two large tubes were passed right down into the bone, and the rest of the wound was lightly packed with gauze.

The whole arrangement of the surgical clinic was that of a very well-equipped modern hospital, with every facility for teaching and research. A lecture theatre in which operations could be performed, a pathological museum, in which the most noteworthy item was a very complete collection of diseases and injuries of the bones and joints, and a department for pathological and clinical research, were some of the most striking features of the surgical unit.

SHORT NOTES OF RARE OR OBSCURE CASES

TORSION OF THE HYDATID OF MORGAGNI

By ALBERT J WALTON, LONDON

MR G H COLT has emphasized the rarity of this condition, and states that in addition to his own recorded case he could only find evidence of the occurrence of one other. The lesion is important, however, in that it closely simulates torsion of the testicle so that an orchidectomy may be wrongly carried out. Of the rarity of the condition there can be no question. Until Mr Colt's case was reported I could find no other recorded, although I had met with a case in my own practice in 1913. The account of this is as follows —

W W, a boy, age 13, was admitted to the London Hospital on March 10, 1913.

He stated that three days before he had noticed a sudden onset of severe pain in the left testicle while at school. He did not vomit, and his bowels were opened regularly. The pain persisted that night. It had since gradually abated, although the left testicle had continued to be very tender.

CONDITION ON ADMISSION—The patient's general condition was good. The left half of the scrotum was œdematous and red, the swelling spreading up to the abdominal wall. The left testicle itself was swollen and very tender, and there was diffuse tenderness of the whole of the cord. The testicle could be felt, and apparently the epididymis was situated in the normal position behind. The right side of the scrotum was not swollen, nor was there any tenderness or swelling of the right testicle or cord. The temperature was 98.6°. There was no evidence of any urethritis and no history of mumps. The abdomen moved well, and it was not distended. There was no tenderness of either abdominal ring, and no impulse could be obtained. There were no enlarged glands in either groin.

OPERATION—An emergency operation was performed in the belief that the condition was one of torsion of the testicle. An incision was made over the left inguinal canal, the external oblique was divided, and the testicle and cord were drawn up into the wound. The cord was much swollen and œdematous.

The tunica vaginalis was dilated and contained a considerable amount of clear fluid through which could be seen a small object, about the size of a currant, which was black or dark blue in colour. The tunica vaginalis was punctured and the fluid collected in a sterile tube. On opening the tunica vaginalis a large, swollen hydatid of Morgagni was seen. It was attached to the outer side of the testicle, and there was a pedicle about a quarter of an inch long which was twisted in two complete revolutions.

The hydatid itself was tense, swollen, and plum-coloured, and about the size of a currant. The pedicle was ligatured and the hydatid removed. There was no mesentery between the testicle and epididymis, and no evidence of torsion of the cord. The tunica vaginalis was sutured, the testicle replaced, and the wound closed.

The wound healed by primary union, and the boy has since had no further trouble. The bacteriological report stated that the fluid from the tunica vaginalis was sterile.

PRIMARY JEJUNAL ULCER

By ALBERT J WALTON, LONDON

THE frequency of gastrojejunal or jejunal ulcer following a gastro enterostomy for a pyloric or duodenal ulcer has directed considerable attention to this portion of the intestine. In spite of the care that is given to the technique in the performance of the operative steps, the frequency remains at or about 2 per cent. In the search for the cause much stress has been laid upon the presence of the acid stomach-contents in the lumen of the intestine, and that this is an important factor is shown by the absence of such a complication after a gastro enterostomy for carcinoma and by its rarity after a similar operation for a lesser-curve ulcer.

The frequency with which unabsorbable sutures have been found in the base of the ulcer leads, however, to the belief that mechanical trauma and errors of technique may also be a predisposing cause.

If these really are causative factors, not only would the complication be expected to occur less frequently as the technique improves, but peptic ulceration should occasionally be discovered in the jejunum apart from operative treatment, for, according to C. A. Roedel¹, contents of marked acidity have been obtained from the distal duodenum by means of a duodenal tube after feeding with a soft mixed meal.

Few such cases have, however, been recorded. In fact, the only case I can discover is one that is reported by Schminsky² and quoted by Judd³. Several other cases may have occurred, but have not yet been reported. The following is such an one—

E. C., a married woman, age 45, and the mother of two children, was seen on Oct. 27, 1919. She stated that she had suffered with stomach trouble for nineteen years. She would have attacks of pain every day which recurred for one to two weeks and then she would remain perfectly free for some months. The pain was situated in the epigastrium, passing to the back and to the whole of the abdomen, and much more to the lower part than is usually seen with a gastric ulcer. The pain would come on late after food and would often be relieved by food. Sometimes it would wake her in the early morning, usually at 2 a.m. There had never been any vomiting. The appetite was good and there had never been any hæmatemesis.

The last attack had commenced five weeks before she was seen, and had continued since. The pain had been more severe in character than in the previous attacks and on the first day it was associated with vomiting. For three weeks she had remained in bed. During this attack she had lost one and a half stone in weight. The bowels had always been constipated, and there had never been any loss of blood or mucus from the bowels.

On physical examination she was found to be a pale and anæmic woman, and looked considerably older than her age. The stomach was not dilated and there was no evidence of ptosis. In the mid-epigastrium point a soft ill defined swelling could be felt which was tender to the touch.

The test-meal revealed free HCl 0.12 per cent, and a total acidity of 50.

The x-ray picture showed the stomach somewhat high, being apparently pushed up by a swelling beneath it. The movements were normal and there was no irregularity in outline. The meal passed freely through the intestine, and the swelling, which appeared to push the stomach up, did not appear to be directly connected with the gut.

The abdomen was opened by an upper right rectus incision, the muscle being displaced outwards. The stomach and duodenum, gall-bladder, and appendix were in every way normal, but on the jejunum about three feet from the duodenojejunal flexure was an inflamed and indurated area about two inches long. Here the wall was injected and thickened, and the omentum was adherent. The mesenteric glands were considerably enlarged. There was no suggestion of growth, and no evidence of miliary tubercle, neither was there any inflamed area to be seen in the rest of the gut. Six inches of the small intestine containing the whole of the inflamed area was resected. The ends of the gut were closed and a lateral anastomosis was performed. The wound was closed.

The gut on section showed a rounded ulcer with a smooth floor and edges in every way comparable with a chronic gastric ulcer

Microscopic section showed chronic inflammatory changes only, with destruction of the mucosa and muscle, there were no changes suggestive of tuberculosis or syphilis

The patient made an uninterrupted recovery, and was discharged from hospital in fourteen days. Before leaving a Wassermann test was taken and was negative

She was seen at regular intervals and continued to have slight ill-defined dyspepsia for about four months. She was troubled considerably with uterine prolapse, and in December 1920 an operation to rectify this was performed by Mr Luker. He tells me he examined the gut at operation and found it had healed perfectly and that there was no trace of any ulceration. Since this time the patient has been seen at regular intervals the last note being dated Feb 3, 1922, and it states that she is remaining entirely free from pain. There is no vomiting, she is feeling well, and gaining weight

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LARGE URETERAL CALCULUS

By P. MAYNARD HEATH, LONDON

THE patient is a male, age 30. When he was 8 years old a calculus was removed from his bladder by suprapubic cystotomy and four years later an operation for the cure of a right inguinal hernia was performed. In 1917, while in France, he noticed that his urine was turbid. In June, 1921, he passed a little bright blood in his urine and suffered a little pain.

On admission to hospital in October, 1921, the man looked healthy. In the left inguinal region above the middle of Poupart's ligament a cylindrical hard tumour could be felt rising out of the pelvis. The kidneys could not be felt. The urine was turbid neutral in reaction, and contained pus. A radiograph showed a large shadow in the



FIG. 123.—Radiograph showing calculus in ureter

region of the pelvic portion of the left ureter (Fig. 123).

On Oct. 5 cystoscopy showed a greatly dilated left ureteral orifice with a small amount of urine escaping from it. The right ureteral orifice was slightly dilated and discharged urine vigorously. No calculus was visible.

The left ureter was then exposed extraperitoneally by a muscle-splitting incision in the left inguinal region. The ureter was very large, and the calculus was easily felt. A longitudinal incision was made in the ureter on to the upper end of the calculus, some turbid urine escaped and was mopped up. The calculus was seized in forceps and coaxed out of the ureter. In so doing the ureter became partially everted owing to the close contact of the ureter and stone. A sound was then passed up to the kidney and down to the bladder. No further calculus was felt, and the wound in the ureter was closed with three layers of catgut sutures. The abdominal wound was closed save for a rubber dam drain. There was a leakage of serum for two days, but healing was complete in ten days. The daily output of urine averaged 50 oz. The amount of pus rapidly diminished, but the reaction remained alkaline. The man was discharged from hospital on Oct 25.

On Dec 25 1921, and again on Jan 7, 1922, the patient had attacks of right renal colic, and on Jan 8 passed a little blood in his urine. On Jan 16 the urine still contained pus. Cystoscopy, after the injection of indigoearmine intravenously, showed a small calculus in the bladder, copious blue efflux from the right ureter, and a flow of pus and faintly blue urine from the left ureter.

The main part of the calculus is cylindrical, but there is a beak-like process at each extremity (Fig 124). The length between most distant points is $3\frac{7}{8}$ in., but by making a model and unfolding the curves the total length is found to be 6 in. The greatest diameter is 1 in. and the weight 1075 gr., or 65.8 gm.

A review of the literature from 1913 to 1921 shows that the largest single ureteral stone is that recorded by Ley. It was 3 in. long, 4 in. in girth, and weighed 995 gr., it was accompanied by pyonephrosis. Other large calculi are described by Specklin, length 11 cm., weight 51 gm., Perrine, length 9.5 cm., weight 41 gm.

Fisher reports a case in which the x-ray picture of the calculus was $4\frac{1}{2}$ in. long. The stone after removal is not described. The kidney was destroyed by supuration and was removed. The case is similar to the one now recorded in that there had been no urinary symptoms, but vague abdominal pains for ten years. For the relief of these the appendix had been removed and a short circuit of the colon carried out.

Abell removed a ureteral calculus, oblong in shape, with a distinct curve or beak at either extremity, thus resembling the one now described. It weighed 24 gm. In the remarkable case recorded by Collinson, there were two calculi forming a cast of the ureter. The upper stone was rather more than 5 in. in length and weighed 840 gr., the lower was $2\frac{1}{2}$ in. long and weighed

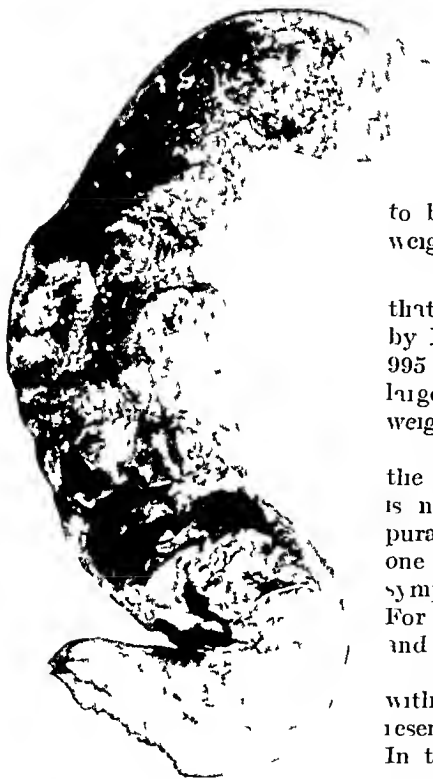


FIG 124.—Photograph of the calculus
(Natural size)

140 gr. The kidney and ureter were removed.

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A CURIOUS HERNIA

By DUNCAN C. L. FITZWILLIAMS, LONDON

THE following curious case of hernia seems well worth reording. A patient, age about 50, had double inguinal hernias which were operated upon in 1913. A year later both had recurred, and he had worn a truss since then. The right side had always been worse than the left, and gave him much trouble, as the truss did not control it.

I first saw him in Petrograd, in 1917, and advised that he should be operated upon again as soon as he got home, as the revolutionary times were not the best to choose for an operation.

In November, 1921, he consulted me again about the hernias, that on the right side being particularly troublesome. I gave him the same advice as before, namely, to have them operated upon as soon as convenient. He told me his truss-maker had strongly advised against operation, and had said that they were best left alone, as the rings were too big. The truss-maker was evidently afraid of losing a client, as the rings were not larger than admitted the end of one finger, and, after all, the larger the ring the more difficult is it to control by a truss. As the hernia was always coming down in spite of the truss, and was becoming obstructed and painful and interfering with his work, an operation seemed to me to be highly necessary.

A few days later I received a wire from him saying that his rupture had strangulated, and asking what he should do.

I wired him at once to call in Dr. Frazer of Cressage and wrote to Dr. Frazer.

Two days later he came up to London for operation, bringing a note from his doctor to say that he had managed to reduce the hernia. The letter warned me that the doctor thought the patient rather nervous and inclined to make much of the affair. In this I agreed, as he could hardly walk, and complained of great pain down the leg, which he said was a severe sciatica that had come on quite suddenly after the doctor had reduced the hernia. It was with difficulty that he could get into a cab at the station.

Up to this time it had always been the right side which had given him so much trouble.

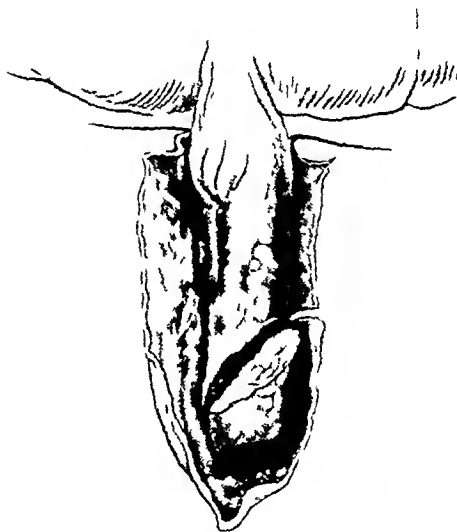


FIG. 12a.—The sigmoid colon above, from which hangs the enormously enlarged and engorged appendix epiploica surrounded by the sac.

but this time it was the left hernia. His bowels, which had not acted for some days, had acted very well after a large dose of castor oil, so there was no obstruction. Locally there was some thickening along the site of the sac, which was tender. This was attributed to the fact that he had recently had the strangulated contents reduced. There was no impulse felt.

At the operation the sac was laid bare, quite black, and obviously containing something, and that something black, strangulated, and dead. The neck of the sac was right up against a piece of large bowel, and the long process in the sac was at first thought to be an appendix, though on the left side of the body. Finding that the longitudinal bands did not enter it, the process was ligated and removed, together with the whole sac. On cutting it open the mass was found to have been an enormously enlarged appendix epiploica, which had been attached to the sigmoid colon (*Fig. 125*). Immediately after the operation it was found that the attack of sciatica had disappeared.

The symptoms were not those of strangulation except for the fact that there was no impulse on coughing. The bowels moved well after the castor oil, and the symptoms of very acute sciatica down the left leg were, of course, the reflected pain from the sigmoid colon. The symptoms, in fact, corresponded exactly to those found both in a Littre's and in a Richter's hernia. This curious form of hernia might justly be classified as a third form of hernia involving the bowel in which the lumen of the bowel is not obstructed. We then should have Littre's hernia, Richter's hernia, and this form of hernia all in the same group.

REVIEWS AND NOTICES OF BOOKS

Tumours Innocent and Malignant, their Clinical Characters and Appropriate Treatment
By SIR JOHN BLAND SUTTON, LL.D., F.R.C.S. Demy 8vo. Pp. 806, with 383 wood engravings. Seventh edition. 1922. London: Cassell & Co., Ltd. 30s.

SIR JOHN BLAND SUTTON'S well known work has now reached its seventh edition. More than that, it is almost thirty years since the first edition was published (1893), and in these circumstances to review the book at this late date may well appear a work of supererogation. None the less we would like to add our tribute to the great merits of this English medical classic, surely one of the best known and most highly appreciated of all modern medical monographs.

No very radical changes have been effected in the new edition, and the total size of the volume remains practically the same. As in former editions, the strength of the book lies in the great personal experience which the author brings to his task, coupled with the easy diction, the wealth of illustration, and the free use which is made throughout of the facts of development and of comparative anatomy and pathology. It is as entertaining as well as an instructive work, a real philosophical treatise on tumours.

In the new edition the subject matter has been divided into five main sections: *Group I*—Tumour diseases of the connective tissues, *Group II*—Tumour diseases of the teeth, *Group III*—Epithelial tumours, *Group IV*—Teratomas and dermoids, and *Group V*—Cysts. The two single chapter sections of 'Endothelioma' and 'Tumours arising from the chorionic villi', present in the sixth edition, have now been incorporated in *Group III*. The former chapter on endothelioma has been broken up. The part which dealt with mixed tumours of the salivary glands has been included in Chapter 36, 'Epithelial tumours of the pancreas and salivary glands', and the subject matter changed in accordance, while the rest of the chapter, much altered, remains under the new title of 'Epithelial tumours of the meninges'. In this connection it may be noted that the author's ban has fallen heavily on endothelioma in general. In the sixth edition the index included twelve references under this title, in the seventh edition there is but one—endothelioma of the choroid (Parsons). The classification of tumours is notoriously difficult, and the author has probably taken the line of least resistance in adopting a simple histological one. Even so, we find it hard to understand why all ovarian tumours, as well as tumours of the ductless glands, should be included in the group of 'Teratomas and dermoids'.

One of the most attractive features of Sir John Bland-Sutton's book is the frequent reference to illustrative cases drawn both from his own practice and from the literature. Most of the chapters conclude with a list of references to the more important original contributions on each of the subjects dealt with, but in several this is lacking. It is, perhaps, not surprising that the omissions should occur chiefly in those sections of the book in which the author himself speaks with the greatest authority.

The number of illustrations remains the same, but some 20 new ones have been inserted in place of a corresponding number removed. Most of the new figures are good, and several are distinct acquisitions, not only *Figs 27 and 28* (multiple exostosis), 165 and 167 (intestinal polyp), and 279 (an enlarged hyppophysis, *in situ*). On the other hand, *Fig 66* in the new edition (melanosis of the colon) is a poor substitute for the much more typical *Fig 69* in the last edition. In general, the illustrations of clinical cases and naked eye specimens are of the very greatest excellence, but the same cannot be said of the microscopic pictures. Many of these could, with advantage, be dispensed with, more especially as no systematic attempt is made to illustrate the morbid histology of new growth.

As is natural in an individualistic monograph, not all the sections and chapters are of equal merit. We would select for special praise the chapters on lipoma, tumours of bone, uterine fibroids, cancer of the breast and uterus, monsters, tumours of the ovary, and hydatid disease. An excellent chapter on adenomyoma uteri is seriously marred by the absence of any reference to the extra-uterine or migratory form of this affection. Even the short paragraph describing a case which invaded the rectum, together with the illustration (*Fig 199*), has been omitted.

In discussing fibrocystic disease of the testis, the fact of the teratomatous nature of these growths is not referred to while Sir James Paget's classical case of enchondroma of the testis is only earned as far as Kinnick and Pigg's reinvestigation. The subsequent careful study of this case by Nicholson, of Guy's, and his conclusion in favour of its teratomatous nature, ought certainly to be referred to. This chapter, in fact, illustrates very well the chief defect in the book.

its pathology in certain sections, is not quite up to date. We hardly think this criticism can be altogether discounted by the title, "Tumours" their clinical characters and appropriate treatment."

Only passing reference is made to the use of rays e.g. in the diagnosis of tumours of bone. Perhaps in future editions (and we hope there may be many more), it may be possible to insert a few typical radiographs, e.g. of an osteoma, a myeloma, and an osteosarcoma.

In the chapter dealing with the causes and treatment of cancer, we note that the use of Coley's fluid in the treatment of inoperable sarcoma is no longer referred to. Yet good results are still attained.

A welcome feature, adding enormously to the usefulness of the book, is the extension of the index from 12 to 22 pages.

This is a book which will be read with pleasure and profit by surgeons and pathologists, students and practitioners alike, and the new edition can but enhance the reputation of the work and of its distinguished author.

La Lithiase Biliaire By A. CHAUFFARD. 1 large 8vo. Pp. 247, with 26 plates. Second edition. 1922. Paris. Masson & Cie. Fr. 20.

In the preface to the new edition, the author excuses the alteration made in the form of the book in view of the fact that the original spirit is maintained. The only real change in form is in expansion of the remarks which appear in the earlier edition on a ray diagnosis of gall stones into a complete chapter, whereby the opinion is emphasized that no case should be operated upon until such a diagnosis has been carried out. The use of rays shows (1) The presence of calculi in about 50 per cent of cases, (2) The presence of a Riedel lobe—which is a sign of gall bladder disease, (3) The presence of adhesions in nearly 50 per cent of the cases. These three points bear on the indications for operative interference which are given later. The errors possible in a ray work of this kind are freely admitted and discussed.

The real spirit of the book centres round the subject of the pathogenesis of gall stones. Even more than in the first edition is the significance of hypercholesterinæmia emphasized. Every argument in favour of this causation speaks against the infective theory. He shows that the view that typhoid infection is causally anterior to gall stones is stultified by a study of cases of disease other than gall stone disease in this regard. The frequency with which typhoid fever preceded such disease is nearly identical with that met with in cases of gall stones. The chief new argument in favour of the importance of hypercholesterinæmia is an ethnological one—that the people of Japan, like those of Japan, seldom suffer from gall stones (and then only from pigment stones) and have a remarkably low cholesterol content in their blood is compared with Europeans. It is clear that if the disease is usually dependent on some other cause than infection, one of the arguments for surgical interference is removed, and the plea for vaccine therapy (which is discussed by Chauffard) is nullified. On the other hand, the undoubted occurrence of infection is allowed for as being a secondary event, which sometimes certainly requires surgical intervention (e.g. for suppurative changes in the gall bladder and adenæmia).

Reference is made to a histological study showing how gall stones can be formed within the mucosa, immediately beneath the epithelium. Drawings are given illustrating the successive steps leading to the production of a calculus lying free within the gall bladder. Freeted calculi are attributed to this mode of origin. Stones which form within the lumen from the beginning are grouped into those due to (a) stasis with excess of cholesterol in the bile, (b) infection, (c) hæmolytic, with secondary infection.

The chapters on diagnosis and on the clinical effects of cholecystitis upon adjacent organs remain as before, and provide an excellent survey of this subject.

The chapter on treatment occasions surprise, inasmuch as there is hardly a single deviation from the views outlined in the first edition. Dietetic and hydro-mineral therapy are given the first position. Treatment at Vichy and Contrexeville are spoken of highly. It is true that Chauffard allows the admittance of operative interference in cases where the ray examination shows the actual presence of stones, but even in cases of impaction in the duct he hesitates about interference because of the high mortality (he quotes 13.15 per cent in complicated cases of this kind). He is evidently hailing between two opinions when he directs his thoughts to the question of emulous change in gall bladder disease though if he applied the same kind of argument to this topic as he does to the question of post typhoid cholecystitis he might not have become so alarmed at the high percentage of carcinoma cases in which stones also occur.

He is also evidently afraid of interfering surgically in elderly persons, in cases where there is arteriosclerosis, in cases of obesity and in cases of emphysema with chronic bronchitis—always, apparently, because the operation would not remove the hypercholesterinæmia factor to which he gives so high a value. Chauffard remarks that the mortality of operations in the early stage, which does not exceed 1 or 2 per cent is little enough, but is large compared with the mortality of hepatic colic. This quotation perhaps exemplifies as well as possible the partial view of a wide subject taken by the author. A whole series of questions come at once to our lips. Is hepatic colic the only danger? Do catastrophes never occur? Is surgery no claim to relieve suffering as well as to save life?

Though throughout this work a great acquaintance is shown with the early clinical symptoms, where Mohrman is accepted as the authority, and though a good average knowledge of the pathological side of the subject is manifest, there is no evidence that cases are studied on the operation table, where the most valuable of all lessons are learnt. The views on surgical treatment, its scope and results, are therefore only those of an interested amateur.

Apart from special points of this kind, the mere fact of there being only a trivial change in this part of the book itself speaks loudly of Chauffard's low esteem for the surgical treatment of this disease.

Traité d'Urologie By G. MARION. Large 8vo. In 2 volumes. With 418 illustrations and 15 coloured plates. Vol. I, pp. 372. Vol. II, pp. 480. 1921. Paris. Masson & Co. 120 fr.

At divers times and in sundry places we have read articles on diseases of the genito-urinary organs by Professor Marion and have been struck with his power of expressing himself in clear and forcible language, for this reason we looked forward with a good deal of pleasure to the perusal of this new book of his. We may say at once that not only have we not been disappointed but that we are filled with admiration at the comprehensive work before us.

The volumes are excellently bound and are handy to hold, the printing is clear though the type is rather small, the numerous illustrations are most beautifully reproduced and are a great help in explaining the text, especially good are the reproductions of the microphotographs, of which there is a great number, they will be a source of genuine delight to every surgeon who has in him the love of pathology. We think it is not too much to say that the treatise is well worth buying on account of the illustrations alone.

The work is a most comprehensive one and includes the anatomy, physiology, pathology, diagnosis, and treatment (including operative) of the genito-urinary organs and their diseases. As one would expect, Professor Marion is thoroughly up to date, and the reader will find most of the recent work in this field discussed in his pages. The book appears to have been most carefully read over and misprints are conspicuous only by their absence.

We feel no doubt that these volumes will at once take their place amongst the standard works of surgery, and we hasten to offer the learned author mille felicitations on his admirable and lucid treatise.

The Practice of Urology: a Surgical Treatise on Genito-urinary Diseases including Syphilis By CHARLES H. CRETWOOD, M.D., LL.D., F.A.C.S. Third edition. Royal 8vo. Pp. x + 840, with 300 illustrations and 9 coloured plates. 1921. London. Baillière, Tindall, & Cox. 12s. net.

The author of this book, in his preface to this edition, states that he still retains the position expounded in the original edition with regard to the problems met with in the domain of urology; this may be defined as the progressive conservative attitude. Whilst thoroughly agreeing with the author that this is the scientific ideal at which one should aim, we think, after reading the book, that the contents are inclined to weigh down the balance on the conservative side.

The volume is well bound and elegantly printed, there are numerous illustrations, most of which are helpful, and the views on urological surgery therein set forth are such as most English surgeons will agree with.

We think, however, that the author's opinions and statements do err in many instances on the conservative side: there is very little in the book that is new, and it is sometimes hardly up to date. If we may give a few instances that have particularly struck us: under the discussion of infections of the kidney by the colon bacillus, there is no mention of the treatment by alkalis, whilst mentioning numerous injections for use in taking pyelographs, the author omits to point out the value of sodium bromide, and the article on the treatment of prostatic hypertrophy is silent on the recent work by German surgeons on the development of anatomy of this condition.

There is an astonishing picture on page 11 (Fig. 4), it is labelled, 'Anatomy of upper and lower urinary tracts (After Poirier)'. In it the renal vein on the left side passes *behind* the aorta on its way to the inferior vena cava, we have not been able to find this figure in the original work, but from the fact that the left renal vein in this illustration joins the inferior vena cava opposite the third lumbar vertebra and runs obliquely downwards instead of transversely, we should imagine that this is a case of an abnormality: there is nothing in the text to indicate that the author considers this to be so, and the illustration, as it stands, is most misleading.

We would suggest that the whole volume, which shows by its comprehensiveness that the author must have spent much labour in its compilation, would be vastly improved if it were carefully read over before another edition is published, there are numerous mistakes in the grammar and punctuation which set the reader's teeth on edge, some of the illustrations have many lines leading out to the side which were undoubtedly intended to be explanatory, but as the author has omitted to letter them and gives no explanation of them in the subscription or in the accompanying text, they fail to enlighten the reader.

Blood Transfusion By GEOFFREY KEYNES, M.A., M.D. Cantab., FRCS Eng. Second Assistant, Surgical Professorial Unit, St Bartholomew's Hospital. Demy 8vo. Pp 166 + viii, with 13 illustrations. 1922. London. Oxford Medical Publications. 8s 6d net.

THIS work gives a connected account of the whole subject of blood transfusion and of the problems arising from it together with practical instructions for performing transfusion by an efficient and simple method.

The medical profession, physicians and surgeons alike, will welcome this book, for blood transfusion is of rapidly growing importance in modern therapeutics, and the subject has hitherto only been represented in the medical literature of this country by isolated communications concerning special points.

The book is a handy, concise exposition of the subject, consisting of seven chapters and an excellent bibliography, which contains references to nearly all the contributions of importance published up to the present time.

Chapter 1 gives in historical sketch, showing that blood transfusion is no new subject, though technical difficulties rendered it almost obsolete until quite recent years. The enormous concentration of thought demanded of the medical profession by the great War gave a tremendous impetus to the solution of these difficulties, which were largely those of (1) Agglutination problems these are dealt with in a very clear and thorough manner in Chapters 4, 5 and 6 (2) Technique in Chapter 7 the principal methods are reviewed, and a simple and efficient technique is fully described. Of the two great problems, the second could scarcely be made easier than it is by the method the author has used so extensively, though we think he has rather overrated the difficulties of giving blood by syringes. Using 100 c.c. syringes it is quite easy for a single operator to give a pint or more of blood with the aid of a nurse to wash the syringes between use.

The problem of how to overcome agglutination also has been materially advanced through blood grouping. An excellent account of the subject is given, but we are only on the threshold of this huge question. As the author points out the possibilities in connection with blood grouping are full reaching and may be found, amongst other things, to have significance in classifying human beings in their relation to disease tendencies.

Chapters 2 and 3 set out the indications for blood transfusion, and we are pleased to note that the author pays particular attention to the value of this as a means of improving a patient's condition prior to operation. In addition, shock and hæmorrhage as well as hæmorrhagic diseases are reviewed in their relation to this subject.

The book is one which all medical men should possess. Blood transfusion is a therapeutic remedy with which no one can afford to be unfamiliar, and the subject could not be dealt with more intelligibly, concisely and practically than it is in this work. Moreover, the references at the end of the book make it easy for those who wish to go more deeply into the subject to gratify their desire.

Zur Hundertjährigen Geschichte der Chirurgischen Universitätsklinik zu Königsberg
1. Pfr. By PROF. DR. MARTIN KIRSCHNER. Roy 8vo. Pp 88, with 37 illustrations and 3 plans. 1922. Berlin. Verlag von Julius Springer. In Germany, M 36. In England, 4 35s.

PROF. MARTIN KIRSCHNER gives a short but interesting account of the development of the Surgical Clinic attached to the Albert University at Königsberg. It began humbly in 1814 with six beds for surgery and twelve for medicine when the Albert University had only six students. It was fortunate in obtaining a succession of Directors many of whose names became household words in surgery—Unger, Seering, Albrecht Wagner, von Bergmann, Schonborn, Mielchev, Heinrich Braun, von Eiselsberg, Garre, Lexer, Payr and Friedrich followed in succession, and raised the reputation of the faculty to a very high pitch of excellence. Prof. Kirschner shows how the advances were made, giving portraits of his predecessors and plans of the buildings from the time the clinic was unable to gain the entire possession of its own buildings until it attained its present well built and its owner was living in it—

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EPONYMS

By SIR D'ARCY POWER, K B E, LONDON

VI SIR JAMES PAGET (continued)

SIR JAMES PAGET read a paper "On a Form of Chronic Inflammation of Bones (Osteitis Deformans)" on November 14, 1876. It is published in the sixtieth volume of the *Medical-Chirurgical Transactions* and immediately attracted a large amount of attention. Packard, Steele, Kirkbride and Elting wrote upon it in the United States, Lannclongue, Marie, Pozzi, Richard, Thebierge, and Joncheray in France, Gaugele and Wollenberg in Germany. But to this day little has been added to Paget's description, and the cause and curative treatment are still unknown.

The paper begins "I hope it will be agreeable to the Society if I make known some of the results of a study of a rare disease of bones.

"The patient (Fig 128) on whom I was able to study it was a gentleman of good family, whose parents and grandparents lived to old age with apparently sound health, and among whose relatives no disease was known to have prevailed. Especially, gout and rheumatism, I was told, were not known among them, but one of his sisters died with chronic cancer of the breast.

"Till 1854, when he was forty-six years old, the patient had no sign of disease, either general or local. He was a tall, thin, well-formed man, father of healthy children, very active in both body and mind. He lived very temperately, could digest, as he said, anything and slept always soundly.

"At forty-six, from no assigned cause, unless it were that he lived in a rather cold and damp place in the North of England, he began to be subject to aching pains in his thighs and legs. They were felt chiefly after active exercise, but were never severe, yet the limbs became less agile or, as he called them, 'less serviceable', and after about a year he noticed that his left shin was misshapen. His general health was, however, quite unaffected.

I first saw this gentleman in 1856, when these things had been observed for about two years. Except that he was very grey and looked rather old for his age, he might have been considered as in perfect health. He walked with full strength and power, but somewhat stiffly. His left tibia (Fig 126), especially in its lower half, was broad, and felt nodular and uneven as if not only itself but its periosteum and the integuments over it were thickened. In a much less degree similar changes could be felt in the lower half of the left femur. This limb was occasionally, but never severely, painful, and there was no tenderness on pressure. Every function appeared well discharged, except that the urine showed rather frequent deposits of lithates. Regarding the case as one of chronic periostitis I advised iodide of potassium and liquor potassæ, but they did no good.

"Three years later I saw the patient with Mr Stanley. He was in the same good general health, but the left tibia had become larger, and had a well-marked anterior curve (Fig 127), as if lengthened while its ends were held in place by their attachments to the unchanged fibula. The left femur also was now distinctly enlarged, and felt tuberculous at the junction of its upper and middle thirds, and was arched forwards and outwards so that he could not bring the left knee into contact with the right. There was also some appearance of widening of the left side of the pelvis, the nates on this side being flattened and lowered, and the great trochanter projecting nearly half an inch further from the middle line. The left limb was about a quarter of an inch shorter than the right. The patient believed that the right side of his skull was enlarged, for his hats had become too tight, but the change was not clearly visible.



FIG 126

"In the next seventeen years of his life I rarely saw him, but the story of his disease, of which I often heard, may be briefly told, and with few dates, for its progress was nearly uniform and very slow. The left femur and tibia became larger heavier, and somewhat more curved. Very slowly those of the right limb followed the same course, till they gained very nearly the same size and shape. The limbs thus became nearly symmetrical in their deformity, the curving of the left being only a little more outward than that of the right. At the same time, or later, the knees became gradually bent, and, as if by rigidity of their fibrous tissues, lost much of their natural range and movement.

"The skull became gradually larger, so that nearly every year, for many years, his hat, and the helmet that he wore as a member of a Yeomanry Corps needed to be enlarged. In 1844 he wore a shako measuring twenty-two and a half inches inside, in 1876 his hat measured twenty-seven and a quarter inches inside. In its enlargement however, the head retained its natural shape and, to the last, looked intellectual, though with some exaggeration.

"The changes of shape and size in both the limbs and the head were arrested, or increased only imperceptibly, in the last three or four years of life.

"The spine very slowly became curved and almost rigid. The whole of the cervical vertebrae and the upper dorsal formed a strong posterior, not angular curve and in anterior curve, of similar shape, was formed by the lower dorsal and lumbar vertebrae. The length of the spine thus seemed lessened and from a height of six feet one inch he sank to about five feet nine inches. At the same time the chest became contracted narrow, flattened laterally deep from before backwards, and the movements of the ribs and of the spine were lessened. There was no complete rigidity, as if by union of bones but all the movements were very restrained, as if by shortening and rigidity of the fibrous connections of the vertebrae and ribs.

"The shape and habitual posture of the patient were thus made strange and peculiar. His head was advanced and lowered, so that the neck was very short and the chin, when he held his head at ease, was more than an inch lower than the top of the sternum. The short narrow chest suddenly widened into a much shorter and broad abdomen, and the pelvis was



FIG 127

wide and low. The arms appeared unnaturally long and though the shoulders were very high, the hands hung low down by the thighs and in front of them. Altogether, the attitude in standing looked simian, strangely in contrast with the large head and handsome features.

' But with all these changes in the shape and mobility of the head, spine, and lower limbs, the upper limbs remained perfect, and there was no disturbance of the general health.

" In 1870, when the disease had existed sixteen years, the left knee-joint was, for a time, actively inflamed, and its cavity was distended with fluid. But the inflammation soon subsided, only leaving the joint stiffer and more bent.

" About this time some signs of insufficiency of the mitral valve were observed, but the patient now lived so quietly, and moved with so little speed, that this defect gave him no considerable distress.

" In December, 1872, sight was partially destroyed by retinal hæmorrhage, first in one eye, then in the other, and at nearly the same time he began to be somewhat deaf. In the summer of 1874 he had frequent cramps in the legs and neuralgic pains, which were described as 'jumping over all the upper part of the body except the head', but change of air seemed to cure them.

" In January, 1876, he began to complain of pain in his left forearm and elbow which, at first, was thought to be neuralgic. But it grew worse, and swelling appeared about the upper third of the radius and increased rapidly, so that, when I saw him in the middle of February, it seemed certain that a firm medullary or osteoid cancerous growth was forming round the radius. After this time there was gradual failure of strength and emaciation, and on March 24 after two days of distress with pleural effusion on the right side he died.

" The body was examined five days after death. As it lay on a flat board its position was remarkable for the head was upraised to the level of the sternum, being supported by the rigid and arched spine, and the lower limbs with the knees bent and stiff rested on the heels and nates.

' The pleura covering the right lung contained small nodular masses of pale cancerous substance and there were many small masses of cancer in the left pleura and in the anterior mediastinum. The upper third of the left radius was involved in a large ovoid mass of pale grey and soft white cancerous substance, similar to that of the nodules in the pleura and mediastinum.

The right femur, the left tibia, the patella and the upper part of the skull were taken for separate examination. In the other bones of the skeleton, except the left radius, no signs of disease appeared externally, but I regret that they were not all more carefully examined for I think that at least in the clavicles and pelvis, some changes like those in the long bones of the lower limbs would have been found.



FIG. 128

Figs 126, 127, and 128 are from photographs of the patient taken about six months before death. Figs 126 and 127 being copied from *Med. Chir. Trans.*, vol. lx.

Sir James then considers in considerable detail the pathological changes which the bones had undergone, and arrives at the conclusion that the bones of the vault of the skull were in every part increased to about four times the natural thickness. In the long bones the periosteum was not visibly changed, not thicker, or more than usually adherent. The outer surface of the walls of the bones was irregularly and finely granular, and everything seemed to indicate a greatly increased quantity of blood in the vessels of the bone. The compact substance of the bones was in every part increased in thickness, the thickening being due to outward expansion and some superficial growth. In some places there were faint appearances of separation of parts of the outer layers of the walls, and

of these becoming thick and porous, while the corresponding parts of the inner layers were less changed, but in the greater part of the walls the whole construction of the bone was altered into a hard, porous, or finely reticulate substance, like very fine coral. In some places, especially in the walls of the femur, there were small ill defined patches of pale, dense, and hard bone, looking as solid as brick (*Fig 129*). Details are then given of four other cases which Sir James had seen or heard of, and which he thought might be similar. The paper concludes with the results of an exhaustive search through the literature and the museum specimens of enlarged bones.

He says "Holding, then, the disease to be an inflammation of bones, I would suggest that, for brief reference, and for the present, it may be called, after its most striking character, *Osteitis deformans*. A better name may be given when more is known of it." No better

name, however, has yet been found for the condition, and when it is not called 'Paget's disease of bone' it is known as '*osteitis deformans*'

Paget's name is also associated with the *haustus hydrargyri perchloridi cum potassii iodido* of the St Bartholomew's Hospital Pharmacopœia. It appears for the first time in the edition of 1882, and has always been known as 'Paget's mixture'. The formula is solution of perchloride of mercury one fluid drachm, iodide of potassium, five grains, compound tincture of cardamoms, twenty minims, and distilled water to one fluid ounce.



FIG. 129.—Sections of the femur, patella, and calvaria from Sir James Paget's case of osteitis deformans. From St Bartholomew's Hospital Museum by kind permission of the Governors.

HYDROCEPHALUS.

By JOHN FRASER AND NORMAN W. DOTT, EDINBURGH

HYDROCEPHALUS has hitherto ranked as one of the most intractable and unpromising of the diseases of childhood. A multitude of surgical procedures has from time to time been instituted for the relief of the condition, but almost without exception they have failed in their purpose, and the rare occasions on which they have succeeded have left one wondering whether, after all, the success was no more than a coincidence—whether, in fact, improvement would have occurred if nothing active had been done, for spontaneous arrest of hydrocephalus is an actual—though rare—possibility. This unsatisfactory state of affairs depended largely upon erroneous ideas regarding the etiological pathology of the disease.

As early as 1862 Hilton,¹ in his *Lectures on Rest and Pain*, described varieties of obstructive hydrocephalus arising from congenital obliteration of the aqueduct of Sylvius and from occlusion of the foramina present in the roof of the fourth ventricle. The clue which Hilton thus gave to the pathology of certain types of the disease does not appear to have produced in the past any operative interference of lasting and definite value, and we owe to Dandy and his co-workers the establishment of operative principles which hold out the prospect, as far as we can at present tell, of permanent cure of the disease. In the course of our work we have had the opportunity of treating a number of children and infants, the victims of hydrocephalus, and since the publication of Dandy's² original papers we have followed up the subject with the closest interest, after four years partly spent in observing and treating these cases we venture to put forward our experiences and our results.

THE NORMAL ANATOMY AND PHYSIOLOGY OF THE PARTS INVOLVED

It is essential to have a proper idea of the processes involved in the production and absorption of the cerebrospinal fluid, and of the relationship of the parts connected with its circulation. Such knowledge is fundamental, because the development of hydrocephalus necessarily entails an error either in the production, circulation, or absorption of the fluid.

The Production of the Cerebrospinal Fluid—

¹ *From the Choroid Plexuses*—We may assume that the bulk of the cerebrospinal fluid is derived from the choroid plexuses which lie in the lateral ventricles. Dandy³ has produced confirmatory evidence of this assumption by experiments on dogs. He occluded the foramen of Monro by a strip of fascia or peritoneum, and a dilatation of the corresponding lateral ventricle resulted. He occluded the iter by means of an oiled gelatin capsule containing cotton wool and a dilatation of the two lateral and the third ventricles ensued. He repeated these experiments, exercising in the first case the corresponding choroid plexus and in the second the choroid plexus of both lateral ventricles. In the first instance the lateral ventricle collapsed completely, in the second, a slight degree of dilatation of the ventricles occurred on account of the remaining choroid plexus of the third ventricle. Absolute proof was thus given that the choroid plexus is the site of production of the cerebrospinal fluid.

The method by which the fluid is actually produced from the villi is uncertain. It is not yet possible to say whether the process is one of secretion, filtration, or distillation. The fluid differs so widely from that secreted by most other glands, and it so persistently retains its freedom from contamination by such body fluids as bile in jaundice, or by drugs

when they are injected into the body, that we must assume that whatever the method of production, certain strong selective actions are at work. The time in embryonic life at which the formation of the fluid first appears remains doubtful, but one anatomical detail would suggest that it is unlikely it is produced in any quantity before the fifth month of intra-uterine life. It is only after the fifth month that the tela choroidea demonstrates the perforations of the foramina of Magendie and Luschka. Therefore any production in quantity of cerebrospinal fluid before the fifth month would have no obvious means of exit from the ventricular system.

2 *From the Perivascular Spaces*—Another possible source of the production of the cerebrospinal fluid exists in the perivascular spaces. Lymphatics, as we generally understand them, do not exist in the brain or meninges, their place is taken by perivascular spaces or channels, and these cannot be grouped as lymphatics because the fluid which they contain has neither the constitution nor the characters of lymph. The perivascular

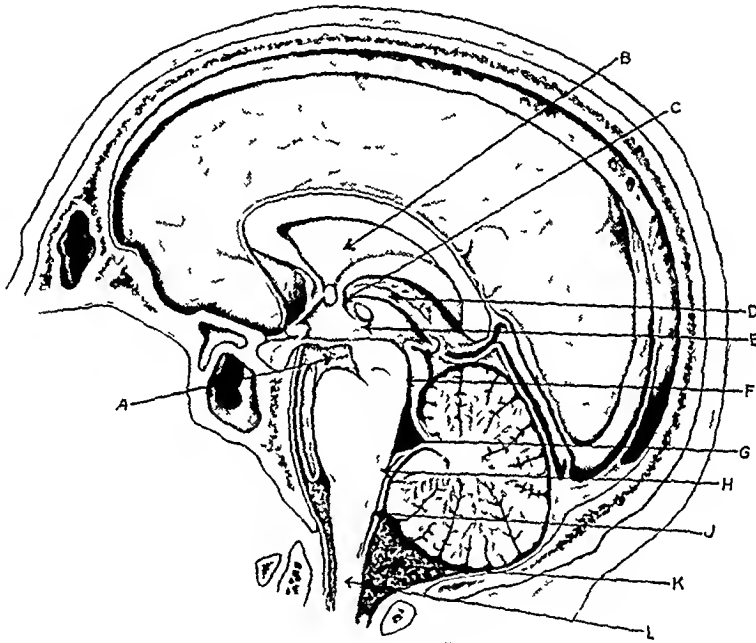


FIG. 139.—Sagittal section through head showing the anatomy of the ventricular system and basal subarachnoid cisterns. (A) Cisterna interpeduncularis. (B) Septum pellucidum. (C) Foramen of Monro. (D) Choroid plexus of third ventricle. (E) Third ventricle. (F) Aqueduct of Sylvius. (G) Fourth ventricle. (H) Choroid plexus of fourth ventricle. (J) Foramen of Magendie. (K) Cisterna magna. (L) Central canal of spinal cord.

spaces extend throughout the cerebral vascular system, even to the finer capillaries (their presence was fully demonstrated by Spina⁴), and it would seem that they contain fluid of simpler constitution than lymph, which passes into them from the blood stream.

The perivascular spaces communicate with the subarachnoid space, and therefore the fluid of the former must to some extent be included in the cerebrospinal fluid. It is an interesting point in the physiology of the central nervous system, though not directly applicable to the subject of hydrocephalus, that the waste products of the central nervous system probably accumulate in the fluid of the perivascular spaces, and are thence conveyed into the subarachnoid space to mix with the cerebrospinal fluid.

The Course of the Fluid after Production—The fluid circulates along the ventricular system and escapes from it through the medium of the central rounded foramen

of Magendie, and the more slit-like lateral foramina of Luschka. It is now in the subarachnoid space, and at first it tends to collect in the large subarachnoid cisterns which lie at the base of the brain (Fig 130), the cisterna magna on the dorsal aspect and the cisterna pontis with its various subdivisions on the ventral aspect. The value of the cisterns is two-fold—they stabilize the pressure of the cerebrospinal fluid, and they render possible a uniform distribution of the fluid in its further progress over the cerebral hemispheres. From the subarachnoid spaces and cisterns the fluid passes in two directions—a small proportion passes downwards through the foramen magnum into the subarachnoid space of the spinal meninges, while the larger proportion escapes upwards between the mid-brain and the tentorium cerebelli to be distributed in the wide area of the subarachnoid space which covers the cerebral hemispheres. It is in this area that absorption of the fluid occurs.

The Method of Absorption of the Fluid —

1 *Pacchonian Bodies*—It was formerly held that the Pacchonian bodies were the media through which the cerebrospinal fluid passed from the subarachnoid spaces into the cerebral sinuses (Fig 131). Recently, however, objection has been raised to this view for two reasons—first, because the Pacchonian bodies are demonstrable only in man and in certain anthropoids, secondly, because in man and anthropoids they are only met with in adult life.

2 *Arachnoid Villi*—Weed⁵ and his collaborators have demonstrated that, while the Pacchonian bodies undoubtedly play a part in absorption of the cerebrospinal fluid, other means exist. Using a Prussian-blue reaction they were able to demonstrate the occurrence of what they have termed 'arachnoid villi'. These are delicate coil-like structures of interlacing strands of connective tissue prolonged from the arachnoid into the walls of the dural sinuses. The Pacchonian bodies are examples of hypertrophied arachnoid villi and this explanation disposes of the two objections already quoted. There is therefore the passage of the cerebrospinal fluid into the bloodstream of the dural sinuses through the medium of the arachnoid villi and, in later life, through hypertrophied examples of these—the Pacchonian bodies. The means by which the cerebrospinal fluid passes from the arachnoid villi into the venous sinuses is a double one—there is a process of filtration from a point of higher to one of lower pressure, and there is a process of osmosis from a fluid of low colloid and crystalline content to one of higher content.

3 *Arachnoid Mesothelial Cells*—Weed⁶ has demonstrated another method by which absorption of the cerebrospinal fluid may occur. Surgeons have recognized that on exposing the outer aspect of the dura (as for example, in such a procedure as an osteoplastic craniotomy) there is often a faint oozing of cerebrospinal fluid through what appear to be tiny pores in the dural structure. Further the blood in this situation has a distinctive watery appearance. Weed believes he has explained these occurrences by demonstrating small nest-like collections of arachnoid mesothelial cells which lie in the dura and are in continuity with the arachnoid villi. Through such channels there is in all probability a slight escape of cerebrospinal fluid on to the outer surface of the dura, where it is absorbed into the circulation.

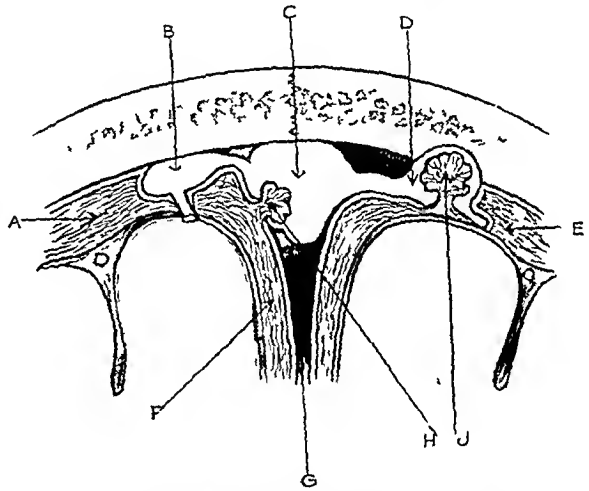


FIG 131—Diagrammatic representation of mechanism of absorption of cerebrospinal fluid showing arachnoid villi connected with subarachnoid space and projecting into a cerebral blood sinus. (A) Subarachnoid space (B) Lacuna lateralis (C) Superior longitudinal sinus (D) Lacuna lateralis (E) Subarachnoid space (F) Subarachnoid space (G) Falx cerebri (H, J) Arachnoid villi (Pacchonian bodies).

4 *Lymphatic Absorption*—An accessory pathway of absorption exists through the medium of the lymphatic system. Key and Retzius were able to inject the cervical lymphatics from the spinal subarachnoid space, and later observers have obtained similar results. The practical importance of this demonstration was not fully appreciated until Weed, using the Prussian-blue method, showed that a subarachnoid injection reached the perineural lymphatics of the cranial nerves and the cervical vessels and glands. Evidently, then, the subarachnoid space is continuous along the perineural spaces of the cranial nerves with the perineural lymphatic channels, and through the medium of this connection an absorption of cerebrospinal fluid must occur.

The Special Anatomy Involved—Of the special anatomy of the parts little need be said. A description of many of the anatomical details is unnecessary in a contribution of this description, therefore only those points which have a direct bearing on the problems of hydrocephalus are mentioned.

The large proportion of the cerebrospinal fluid is formed within the lateral ventricles in which lies the bulk of the choroid plexus. The fluid passes by the foramina of Monro into the narrow cleft-like space which separates the mesial surfaces of the thalami—the third ventricle. This ventricle contains a choroid plexus, and therefore it is the site of production of a certain amount of cerebrospinal fluid.

The fluid leaves the third ventricle by the Sylvian aqueduct to pass into the fourth ventricle. The lumen of the Sylvian aqueduct is somewhat narrowed at its two extremities—a point of importance we shall allude to later in connection with congenital obstructive hydrocephalus. In regard to the fourth ventricle there are certain details in the roof of the ventricle which require mention, as they have a close bearing on the subject under discussion. Viewed in median sagittal section (see Fig 130), the roof of the ventricle appears as a tent-like structure, the wings of which, where they come together, bound the space the recessus tecti, which penetrates the cerebellar medulla between the superior and inferior vermes. The upper wing of the tent is formed by the 'superior medullary velum' connected with the corpora quadrigemina above, the cerebellar medulla below and the superior cerebellar peduncles laterally.

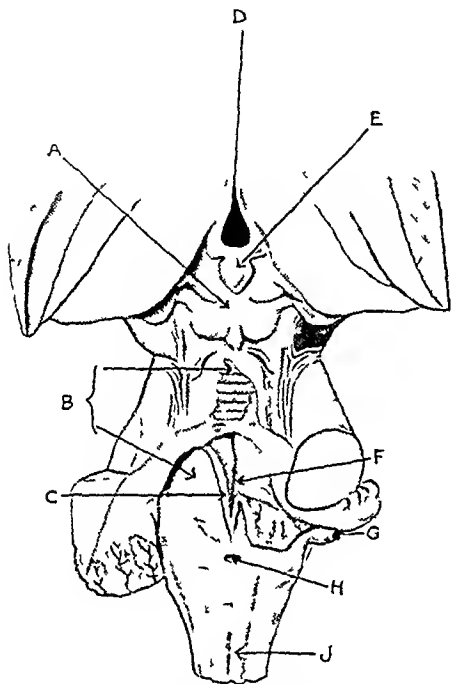


FIG 132—Dissection exposing the roof of the fourth ventricle. The lower part of the roof has been partly opened and turned down. (A) Foramina quadrigemina (B) Roof of fourth ventricle (C) Choroid plexus of fourth ventricle (D) Third ventricle (E) Pineal body (F) Floor of fourth ventricle (G) Foramen of Magendie (H) Foramen of Luschka (J) Medulla oblongata

It is, however, in the lower wing of the tent that, from our point of view, the chief interest lies (Fig 132). It is composed of two parts, an upper thicker crescentic plate of white matter (the inferior medullary velum) and a lower extremely thin membrane (the tela choroidea). The latter structure is formed in a morphological sense of ependyma only, though actually it is supported by a backing of pial tissue. During the early part of foetal life the tela choroidea is a complete membrane, but about the fifth month it becomes perforated at its lower extremity by an aperture which remains throughout life, the foramen of Magendie. About the same time two additional clefts (the foramina of Luschka) appear at the lateral extremities of the tela, behind the upper roots of the ninth nerve in the pouch-like extension of the ventricle beneath the flocculus and through these three apertures and probably through them alone the system of ventricular cavities and the central canal of the spinal cord are brought into communication with the subarachnoid space.

Three groups of choroid plexus appear in the roof of the fourth ventricle—a medial and two lateral—and therefore a proportion of the cerebrospinal fluid is formed within this space

Having escaped from the fourth ventricle into the subarachnoid space by the foramina of Magendie and Luschka, the fluid is free to pass forwards over the hemispheres or caudally into the spinal subarachnoid space. We have elsewhere described the absorption of the fluid from the cranial subarachnoid space and we are therefore now in a position to appreciate the cycle of the fluid from its point of production to the area of its resorption into the blood-stream of the various sinuses

THE ESSENTIAL ERROR IN THE FORMATION OF A HYDROCEPHALUS

By the term hydrocephalus we mean an accumulation of cerebrospinal fluid within the cavity of the skull, and, keeping in view the outline which we have given of the formation, circulation, and absorption of the cerebrospinal fluid, we may systematize the possible origins of the disease as follows: (a) *It may be due to an excessive production of fluid*, (b) *It may be due to an obstruction in some portion of the route along which the fluid circulates*, (c) *The production and circulation of the fluid remaining normal, there may be an interference with the absorption of the fluid*

Based upon these three possibilities, a reasonable and scientific classification of the disease can be made, which will include moreover all varieties of the disease

VARIETIES OF CLASSIFICATION

Hitherto different varieties of hydrocephalus were recognized in describing the disease. For example, hydrocephalus was classified as 'acute' or 'chronic', according to the rapidity with which the fluid accumulated, or dependent on its association with coincident acute inflammatory reaction in the meningeal structures. Hydrocephalus was spoken of as 'internal' or 'external' according to whether the fluid accumulated within the ventricular system or in the extracerebral tissue of the arachnoid spaces. Further the disease was classified as 'congenital' or 'acquired', according to the history of its incidence and occurrence. Such an irregular and flimsy classification had no proper basis. The occurrence of an external hydrocephalus is a primary condition distinct from a co-existing internal hydrocephalus has been questioned and its occurrence is universally accepted is a very rare condition. In our personal experience we have seen two examples of it. Both were cases in which a marked degree of hydrocephalus was present at birth and in both cases life persisted for only a few hours after birth. In the two instances a similar condition was found (Fig 133) there was a congenital absence of the quadrigeminal plate and the superior cerebellar peduncles

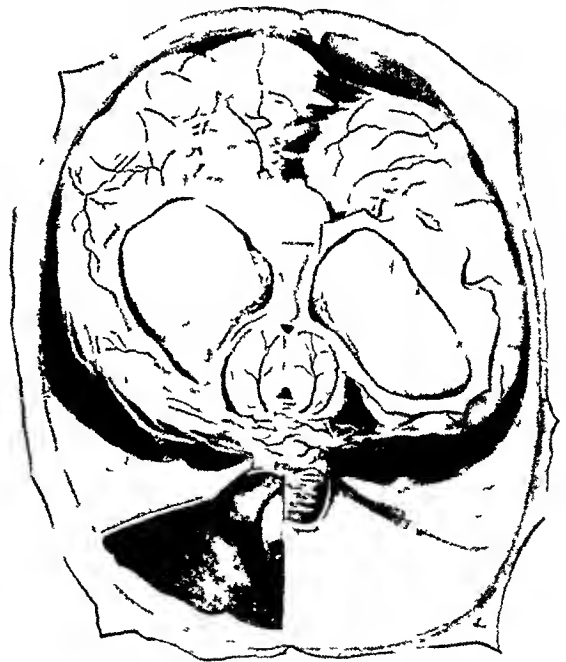


FIG 133—Drawn from a specimen of congenital (developmental) hydrocephalus. The brain and skull are grossly deformed. The upper part of the quadrigeminal plate is absent and the open ends of the Sylvian aqueduct are seen. The cerebral tissue, the roof of the lateral ventricles is defective, so that these cavities open on to the dorsal surface of the brain. A rupture of the membrane of attenuated cerebral tissue attached to the right defect in the extreme ventricular distention at an earlier period. The posterior pole of the skull is disproportionately small and shallow.

was a congenital absence of the quadrigeminal plate and the superior cerebellar peduncles

The posterior wall of the Sylvian aqueduct was absent and the cerebrospinal fluid which was produced within the ventricles was poured directly into the sub meningeal space. The result was that the cerebral tissues were atrophied and ill developed, and displaced forwards into the frontal regions of the cranium, the greater portion of the skull being occupied by cerebrospinal fluid. The condition, therefore approximated as closely as possible to a pure external hydrocephalus, but, even in this instance, there was to some extent a co-existing, though slight, internal hydrocephalus.

The classification of 'acute' and 'chronic' hydrocephalus was specially unfortunate. Apparently in the minds of many observers acute hydrocephalus was synonymous with the development of a basal meningitis. But, whatever the origin of the condition, all varieties of hydrocephalus are chronic in their formation, though the actual period of time involved may vary within wide limits.

Something is to be said for the method of classification into 'congenital' and 'acquired' varieties of the disease—a considerable proportion of cases can without much difficulty be grouped into one or other of these two classes—but the independent use of the two terms is not sufficiently descriptive of the condition of affairs.

The Classification Suggested by Dandy—Recognizing the unsatisfactory condition of the classification nomenclature, Dandy⁷ has suggested that the following is a scientific and inclusive grouping of the possibilities —

Hydrocephalus due to	{	Diminished absorption of cerebro spinal fluid	{	1 Communicating hydrocephalus (due to adhesions in the subarachnoid space)
				2 Obstructive hydrocephalus— Congenital atresia adhesions, tumours
	{	Increased production of cerebro spinal fluid	{	3 External hydrocephalus ⁸
				1 Acute hydrocephalus (increased fluid from inflammatory products in acute meningitis and trauma)
				2 Communicating hydrocephalus (due to occlusions of the vena magna Galeni)

This classification is complete in so far as it would appear to include all varieties of hydrocephalus. From the point of view of the pediatric surgeon, however, it includes types of the disease which, if they actually exist as clinical entities, have little or no relationship to surgical treatment. Under this criticism we include external hydrocephalus, acute hydrocephalus, and communicating hydrocephalus due to occlusion of the vein of Galen.

We have stated our experience with external hydrocephalus, we do not believe that acute hydrocephalus exists as a condition calling for surgical interference. And, while we realize that a non-obstructive variety of hydrocephalus said to be due to occlusion of the vein of Galen, has been produced experimentally (Dandy⁸), its clinical existence is exceedingly doubtful, and it certainly can be accepted as negligible in our estimation of hydrocephalus work.

Excluding, therefore, for practical purposes these three varieties, only the congenital anomalies and communicating and obstructive types (Dandy) remain to be considered.

We would further venture to criticize the title of 'communicating hydrocephalus' (as contrasted with 'obstructive hydrocephalus') as being insufficiently expressive of the condition, or even misleading. According to Dandy, the 'communicating' variety depends upon pia-arachnoid adhesions. The normal filmy pia-arachnoid tissue is replaced by a firm, fibrous, adherent membrane, and further, it is the distribution and location of these adhesions, not their extent, which determine the production of hydrocephalus. Adhesions encircling the mid-brain where it passes through the mesura tentori interrupt the subarachnoid communication between the posterior and middle cranial fossae, and thereby eliminate the cerebral subarachnoid space (the main area of absorption) from participation in the absorption of the fluid. Adhesions which obliterate the cisterna magna or those at the base of the brain will produce hydrocephalus as effectively. If this reasoning is correct, 'communicating hydrocephalus' is as much an example of an obstructive lesion as the intracerebral types of obstruction.

Returning, then, to our original conception of the possible causes of the disease, our experience leads us to the conclusion that increased formation of fluid may be excluded as of little practical importance, and that deficient absorption is not known to occur *per se*. Therefore, for practical purposes we are only concerned with hydrocephalus which is due to congenital anomalies or to an obstruction to the circulation of the cerebro-spinal fluid. The latter type is naturally divided into two main groups in which the obstructive lesion is ventricular or extraventricular. The ventricular group is again divided into degrees according to the exact anatomical site of the lesion.

A More Suitable Classification—On the grounds above stated we suggest the following as a more suitable classification—

GROUP		DEGREE	
		(Site of obstruction)	
Hydrocephalus due to	Congenital anomalies		
	Ventricular (obstruction)	1	Between one lateral and 3rd ventricle
	Extraventricular (obstruction)	2	Between both lateral ventricles and 3rd ventricle
		3	Between 3rd and 4th ventricles
		4	In the roof of 4th ventricle
		5	In the subarachnoid space

The above we submit, is a simple, expressive, and accurate classification from the anatomical and physiological points of view. It is also very significant practically, for, as will appear later, each type has its distinctive and appropriate treatment.

In return use we omit the term 'obstructive' entirely, and we speak, for example, of a ventricular hydrocephalus of the fourth degree, or of an extraventricular hydrocephalus.

THE ETIOLOGICAL PATHOLOGY OF HYDROCEPHALUS

It is important to explain that the conclusions arrived at have been based entirely upon the observations made on the 21 cases under review, with the addition of certain other cases which through the kindness of our colleagues, we have had an opportunity of studying.

1 Congenital Hydrocephalus and the Conditions under which it is met—Under two varieties of conditions hydrocephalus has been met with at birth, and therefore the varieties may be described as examples of congenital types.

a True Congenital Hydrocephalus—There is a well developed form in which the head is considerably enlarged at birth, so much so, sometimes, as to constitute an obstruction to delivery. This variety we have found to be associated with an error in the development of the central nervous system which permits an escape of fluid from an open Sylvian aqueduct. The resulting hydrocephalus is of an external character, though there may be an associated dilatation of the ventricular spaces. It is not amenable to surgical treatment and it is rarely compatible with post-natal life for any prolonged period.

b The Variety of Hydrocephalus which Accompanies a Spina Bifida—At birth attention is naturally directed to the spinal error and the head condition may be overlooked, but we have satisfied ourselves that a certain proportion of spina bifida cases are accompanied by examples of congenital hydrocephalus inasmuch as there is a ventricular dilatation present at birth. We have confirmed this observation by ventriculography during life and by post mortem examination. If the spina bifida is operated on and the defect closed, the pre-existing hydrocephalus soon forces itself on one's attention by the rapid increase in the size of the head. This variety of hydrocephalus is of the extraventricular type in so far as there is no obstruction to the circulation of the fluid up to the point of its exit from the fourth ventricle.

These varieties of hydrocephalus (*a* and *b*) are the only ones which we believe to be congenital and both of them are closely related to an error in the development of the central nervous system.

2 The Origin of Ventricular Hydrocephalus —

a Tumours as a Cause — In a class by itself comes that group which owes the development of the hydrocephalus to the presence of a cerebral tumour. Theoretically a tumour which from its position presses upon any portion of the interventricular system may give rise to a hydrocephalus, practically, it is the cerebellopontine angle which is the usual situation of the neoplasm (*Fig 134*). Its pressure is exerted upon the lower portion of the



FIG 134 — Drawing from a specimen from a case of secondary hydrocephalus. The condition developed secondarily to a cerebellar tumour which occluded the lower part of the Sylvian duct thus leading to a ventricular hydrocephalus of the third degree.

Sylvian aqueduct, and there is a resulting dilatation of the ventricular system above this level. Though in a very real way the hydrocephalus is a secondary development to the tumour, it is interesting that, in the examples of this condition which came under our care, the hydrocephalic condition clinically overshadowed the local tumour formation, and the clinical features which impressed themselves on the examiner were those of the ventricular hydrocephalus rather than the signs of the neoplastic condition.

As we have said, this type of obstructive hydrocephalus stands in a group by itself, and surgical interference is primarily directed towards the tumour—it does not enter into a contribution of this description. There remains a large group of cases of ventricular hydrocephalus for which other causes must be found. Atresiae and adhesions constitute the remaining causal factors.

By the term 'atresia' we mean a narrowing or obliteration of a portion of the interventricular system, while by the use of the term 'adhesions' we imply that adhesions have formed at the base of the brain so as either to occlude the foramina in the roof of the fourth ventricle or to limit the circulation of the fluid in its immediate neighbourhood. From the etiological point of view both of these types have a common basis in so far as both varieties have an inflammatory origin, plastic or infective. We have not met with an atresia which could be accepted as an actual congenital error in the cerebral development—this is an important consideration. There is a wide variety of factors which may lead to the formation of adhesions, and certain causes are apparently more liable than others to produce the errors.

b Birth Haemorrhage as a Cause—

When investigating the case-histories it may be found that the birth was a difficult one, that forceps were employed, and that a good deal of trouble was experienced in the delivery of the head. In fact, in certain cases there was the likelihood that an intracranial haemorrhage had occurred. In seven cases of the series under consideration a natural history of this sort was obtained, and the subsequent operative interference revealed the presence of a haemorrhagic effusion in the membranes at the base of the brain, in certain of these the haemorrhagic effusion was definitely invading the roof of the fourth ventricle, and it could be demonstrated that there was a resulting occlusion of the foramina normally present in the ventricle roof (Fig 135). It would appear that a basal haemorrhage of this description is more likely to occur when the forceps blades are applied in the sagittal axis of the head than in the coronal axis. It seems reasonable that the compression of a forceps blade beneath the occiput is more likely to produce a subtentorial haemorrhage than forceps applied laterally.



FIG 135.—Drawing from a specimen of ventricular hydrocephalus of the fourth degree. The condition developed as the result of a basal birth haemorrhage. There is a haemorrhagic effusion in the external magna which has occluded the foramina in the roof of the fourth ventricle.

We possess very definite evidence that an intracranial haemorrhage produced at birth is liable to be followed by a hydrocephalus if the location of the haemorrhage is subtentorial in its distribution. These remarks apply at this stage only to ventricular hydrocephalus, but we shall allude to the subject again in relation to the extraventricular type.

*c Infective Meningeal Conditions as a Cause—*It is possible that we underestimate the degree of occurrence of infective meningeal conditions in the early period of life—the imperfect ossification of the base of the skull may explain a greater liability to infection from the extracranial regions at this stage than in later life. At any rate, during infancy there is a definite proportion of children who suffer from inflammatory meningeal conditions. The infections are not necessarily fatal, some of them are so slight as scarcely to elicit close attention, more especially as they may be overshadowed by more urgent general symptoms, yet the congestion and effusion which they have induced may be sufficient to give rise to an occlusion of the route of the ventricular circulation, and subsequently to hydrocephalus.

An interesting example illustrative of this point is borne out by the following case-history —

Case 1 — J S (6½ months) About six weeks before admission to hospital the baby had been much in contact with a number of cats who were suffering severely from distemper. The child took an illness which closely resembled the distemper of animals—temperature, coryza, discharge from nose and eyes. The child's uncle is an authority on veterinary matters, and he had no doubt that the child's illness was a direct infection from the animal source. Within ten days of the onset of the illness the child developed what clinically resembled a mild meningitis, with head retraction, vomiting, and eye squint. These symptoms lasted a few days, and a complete recovery then seemed to follow. About two weeks later it was noticed that the head was beginning to enlarge, and a hydrocephalus rapidly developed.

Investigation showed the hydrocephalus to be an obstructive one due to occlusion of the foramina in the roof of the fourth ventricle. So rapid was the enlargement of the head that operative interference was impossible. The child died and subsequent examination demonstrated signs of a former basal meningitis, which had resulted in occlusion of the ventricular foramina.

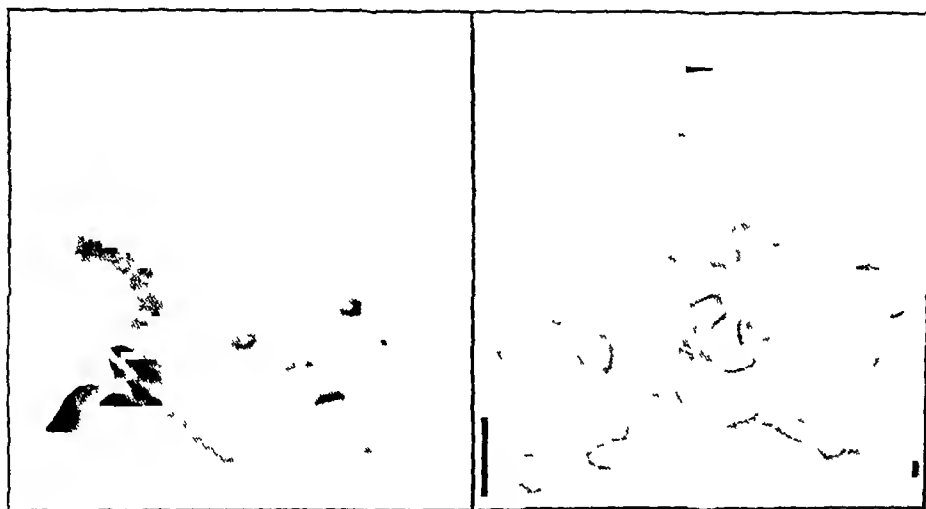


FIG 136—Two examples of syphilitic hydrocephalus. Both cases were ventricular in type.

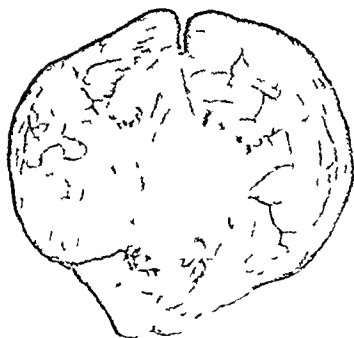


FIG 137—Drawing from a specimen of syphilitic basal meningitis. The condition has led to closure of the foramina of Magendie and Luschka and to a resulting ventricular hydrocephalus of the fourth degree.

In certain cases of the series under consideration we believe that a former subacute basal meningitis was the essential causative factor.

d Congenital Syphilis as a Cause—In certain cases of congenital syphilis there may be an associated chronic inflammatory thickening of the cerebral meninges (Fig 136). Such a change is most marked at the base of the brain (Fig 137), and the more vascular pia arachnoid is affected to a greater degree than the less vascular dura mater. It is in the tissues around the cisterna magna and the cisterna basalis that the plastic and adhesive specific meningitis may be most evident. We confess we have been surprised to find what a relatively large proportion of hydrocephalus are victims of congenital syphilis and show a positive Wassermann reaction. The meningeal changes may develop in the child who otherwise does not show the general stigmata

of the disease—in fact, it would appear that it is in the ill-defined and indefinite types of the disease that the meningeal changes are most in evidence.

It has further been our experience that in the atresic forms of hydrocephalus, where

the obstruction to the circulation of the cerebrospinal fluid is situated above the point of exit from the fourth ventricle congenital syphilis is such a common concurrent condition that we believe that a considerable proportion of the atresiae are specific in origin. The point is one of considerable importance, because in certain cases in which the origin of the hydrocephalus was recognized as being specific we were able to arrest the progress of the disease by means of antisiphilitic remedies without operative interference.

We believe that the possible origins of ventricular hydrocephalus may be summarized into three classes—according to whether it is dependent upon intracranial birth hemorrhage, former subacute inflammatory meningeal condition, or the leptomeningitis which develops in association with congenital syphilis.

3 The Etiology of Extraventricular Hydrocephalus—Until a recent publication of Dandy's work no satisfying explanation had been offered of the origin of the extraventricular type of hydrocephalus. It is clear that no obstruction exists up to and including the points of exit of the fluid from the fourth ventricle. The possibility that there is an over-production of cerebrospinal fluid has never been established, and, if this is not the fault, the only other possible explanation is a diminished absorption of the cerebrospinal fluid. The difficulty has been to explain the obstacle which exists to the absorption of the fluid.

Dandy's view as to the anatomical lesion responsible for this type has already been given, viz., pia-arachnoid adhesions which shut off the absorbing area of the cerebral subarachnoid space from the posterior cranial fossa*. In our series of cases, six have been examples of the extraventricular variety, and we have had an opportunity of satisfying ourselves that the explanation brought forward by Dandy has a great deal to recommend it. Certainly in these cases there is an extensive obliteration of the subarachnoid space by adhesions. They are best illustrated at autopsy, and, to demonstrate them efficiently, the brain should be removed with the dural covering, if possible, intact. From the base of the child's skull the dura can be removed with greater facility than is the case in the adult, and subsequent dissection will then demonstrate that the normal pia-arachnoid membrane is replaced in these cases by an opaque adherent membrane which extends over the region of the cisterna magna and basalis, around the cerebellar hemispheres, and downwards to the foramen magnum. Adhesions in this region must constitute a very real obstruction to the circulating cerebrospinal fluid, and, once the backward pressure has caused reaction and internal hydrocephalus has begun, a vicious circle is actually in existence, because, as the cerebral distention increases, the close contact of the cerebral tissues with the meninges and skull, and, more especially, with the unyielding base of the skull, further increases the obstruction to the upward circulation of the cerebrospinal fluid. The actual origin of the adhesions in extraventricular hydrocephalus is similar to that already discussed in relation to the ventricular type, with the proviso that birth hemorrhages probably do not play a part in the first-mentioned variety. Basal meningitis, simple or specific, is the more likely origin.

SOME POINTS IN THE MORBID ANATOMY OF HYDROCEPHALUS

We have dealt with the more important points in the etiological pathology of the condition, and it remains to review the more outstanding characteristics of the morbid anatomy. The essential feature is a distention of the ventricular system with cerebrospinal fluid. The distribution of the distention will necessarily depend on the type of hydrocephalus. In the extraventricular type the complete ventricular system is involved; in the ventricular variety the distention will depend on the situation of the obstruction.

* Dandy states that he has produced communicating hydrocephalus experimentally by encircling the mid brain with a strip of gauze saturated in iodine, and so inducing adhesions. *Ann. of Surg.*, 1919, lxx, 129.

The following table shows the distribution which has come under our notice

Table 1—THE TYPES OF HYDROCEPHALUS IN THE SERIES UNDER REVIEW

Extraventricular type	6 cases
Ventricular type, 1st degree	0
2nd	0
3rd	7
4th	8
Total	21 cases

(including 2 tumour cases)

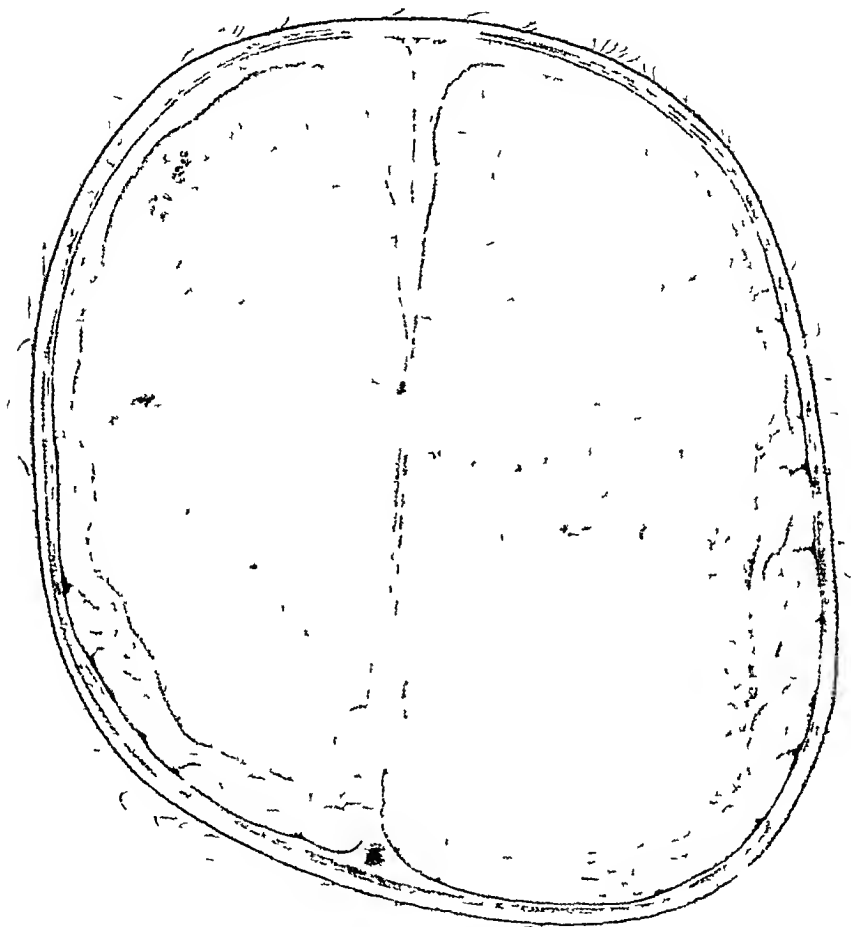


FIG 138.—A frozen section through the head of a ventricular hydrocephalus. It illustrates the extreme degree of destruction of the white matter (Dr Thomson's case)

Our experience has been that the ventricular type is more common than the extraventricular type in a proportion of about 3 to 1, and of the ventricular type the fourth degree of obstruction i.e. in the roof of the fourth ventricle, is the most common in a proportion of 8 cases out of 15.

The increasing distention of the ventricular cavities leads to a progressive thinning of the cerebral tissues, and it is interesting that the disappearance of the cerebral substance is at the expense of the white tissue—even in the most advanced example of the disease a zone of grey tissue of approximately normal thickness exists (Fig 138). The cerebral

sulci become opened out, and sometimes they disappear, thus change is most marked in the frontal region of the brain. The fontanelles, the sutures, and the imperfect ossification generally of the cranial vault encourage an enlargement of the dome of the skull, while the more unyielding base is not correspondingly affected. It is this disproportion which gives the characteristic clinical appearance of hydrocephalus, and which also explains the unlikelihood of there being much displacement of the medulla downwards into the foramen magnum. We shall allude again to the importance of dealing with these cases before the destruction of cerebral tissues becomes marked, the whole basis of successful treatment will depend on its early inauguration. As long as the basal ganglia remain intact life continues, and, as the distention follows the line of the yielding skull in an upward direction, these ganglia are wonderfully preserved from pressure until a late stage, but, from the point of view of making operative interference worth while, close attention should be paid to the degree of destruction of cerebral white tissue which is proceeding.

A puzzling point in pathology is opened up when we attempt an explanation of a variety of hydrocephalus which is accompanied by a complete occlusion of the ventricular system, and yet the hydrocephalus has been spontaneously arrested. Through the kindness of a colleague we have had an opportunity of examining the case-records of two examples of this puzzling condition. The first and the more impressive case was that of a young adult, age 25 years who had died suddenly from what was thought to be a cerebral hemorrhage. There had been a complaint of giddiness and headache with some vomiting. Two hours later unconsciousness supervened, the breathing became stertorous, and within a short period he was dead. In the previous history there was no definite account of serious illness, he had been recognized as a delicate man, nervous and highly strung, constantly subject to headaches and attacks of migraine. The head was not unusually large, and on superficial examination there was nothing to suggest a hydrocephalus. On autopsy a peculiar state of affairs was discovered. The convolutions were flattened and relatively few in number, the entire ventricular system was dilated, and the roof of the fourth ventricle was completely occluded by a dense fibrous membrane. No trace could be discovered of the normal foramina, and tests applied showed that the intraventricular fluid had apparently no communication with the subarachnoid fluid along the usually recognized channels.

The second case occurred in a boy, age 8 years. We have no knowledge of the previous clinical history but the autopsy specimen which we were permitted to examine showed a long-standing occlusion in the roof of the fourth ventricle, which, as far as we could discover, must have completely isolated the intraventricular from the extraventricular fluid.

The importance of cases such as these is that they would appear to indicate one of two possibilities—

1. Either there are intraventricular media for the absorption of cerebrospinal fluid with which we are at present unacquainted, or

2. Under certain conditions the isolation of the intraventricular fluid by occlusion of the foramina leads to a greatly diminished production of cerebrospinal fluid, and further it would seem to presuppose that there are extraventricular sources of production of cerebrospinal fluid for in both of the above cases cerebrospinal fluid was present in the subarachnoid spaces.

It is clear, at any rate, that cases such as these indicate that there are possibilities in the physiology, or at least in the pathological physiology, of the cerebrospinal fluid circulation with which at present we are not completely acquainted.

There are many other details in the pathology of hydrocephalus to which we have not alluded, we have purposely dealt only with those which have a bearing upon the surgical treatment of the disease.

CERTAIN CLINICAL FEATURES OF THE DISEASE

Question of Sex and Age.—There is nothing to be learned from a consideration of the sex. In the series under review there was a curious similarity in the sex occurrence, the figures being respectively 11 males and 10 females.

The age at which the cases came under surgical notice is shown in the following table —

Table 2—THE AGE OCCURRENCE

Age	MONTHS												YEARS			
	1	2	3	4	5	6	7	8	9	10	11	12	2	3	4	5
Cases	—	2	3	5	1	1	2	1	2	—	—	1	1	2	—	—

The majority (18) of the cases were under one year old, 1 child was a year and a half, and 2 of the cases were between two years and three. No cases appeared at a later age than three years.

The Clinical History—In certain cases the statement was offered that the parents noticed the head to be somewhat enlarged at birth, but it is doubtful if any real weight can be attached to this observation. It is clear that in no case was the head of such a size at birth as to offer any difficulty to delivery. There was no case, therefore, which would justify the qualification of being congenital in the sense that the signs of the disease were definitely present at birth. The two examples of true congenital hydrocephalus which have been mentioned are not included in this summary.

In certain instances it was remarked that the scalp veins were unusually prominent, and this feature was observed before any definite cranial enlargement was apparent.

As the head increases in size, the enlargement at first is a gradual one, but after a definite point is reached the distention of the head progresses more rapidly. Dandy¹⁰ offers an observation which appears to contradict this statement, for he believes that the production of cerebrospinal fluid diminishes as the pressure within the ventricles increases, but clinically we are convinced of the truth of the point which we have made. For example, such an observation as this was repeatedly made —

E. H.—

First observation	April 10,	circumference of head	17½ inches
Second	May 12		18
Third	June 15		18½
Fourth	July 10		21½
Fifth	July 30		23

During the last six weeks of observation the head increased by 4½ inches, as compared with an increase of one inch during the first two months of observation. It would seem that, after a definite point is reached, the tension exerted by the skull is so reduced that the underlying cerebral distention is less restricted than before. There is no clinical evidence that there is a diminished production of cerebrospinal fluid as the tension increases.

As the head enlarges the axis of the eyes is displaced downward, so that the sclerotics appear constantly underneath the upper lids. There is often a well-marked strabismus. The occurrence of nystagmus is an unusual feature. In the later stages of the disease sluggishness of the pupils and atrophy of the optic nerve are sometimes present. The mentality of the children ranges between extreme idiocy and normal intelligence. Most of the patients are feeble-minded and apathetic, if old enough, they have difficulty in speaking. Motor disturbances are usually well-marked, and manifest themselves in spasms, paresis, unusual rigidity, and tremors, the lower extremities are more severely affected than the upper. Sometimes the spasms and paresis are more marked on one side than on the other. The reflexes are increased. Twitching of individual groups of muscles and general convulsions may occur. Hydrocephalic children usually present a delicate appearance; they are pale and emaciated with a senile expression. On account of the weight of the head its support is difficult, and therefore it is often bent forwards or thrown backwards. These children generally cry a good deal, and they do not readily put on weight. The appetite is not bad, but digestion is usually retarded.

It is sometimes remarkable how few general symptoms may exist in a hydrocephalus of the most marked degree. The infant a section through whose head is illustrated in Fig 138 was one of this type. It can be recognized how very marked was the atrophy of nerve-tissue, and yet, to within forty-eight hours of death, the intelligence was such that the child recognized its mother, while there was very little evidence of paralysis or rigidity of the superficial musculature.

THE PHYSICAL EXAMINATION OF THE CASE

In the majority of cases it is sufficiently obvious that a hydrocephalus is present but further examination is required if the case under review is to be efficiently classified and efficient classification necessarily involves that an attempt be made to answer three questions. 1 *Does any evidence exist which would indicate the possible origin of the disease?* 2 *To what variety of hydrocephalus does it belong? Is it ventricular or extraventricular in type?* 3 *If it is ventricular in type, at what level does the lesion exist which is responsible for the hydrocephalus?*

The physical examination is directed towards supplying an answer to each of these individual queries so far as is possible. The answers having been given, classification is possible, and an intelligent treatment may be planned.

1 **The Origin of the Disease**—If this question can be answered, the information will be obtained by careful questioning of the parents, a full case-history, and general examination of the child. Attention is specially paid to three possibilities—the occurrence of a syphilitic infection, the infliction of an injury to the skull, such as may have produced an intracranial hemorrhage, and the history of a previous meningeal or encephalic infection. A fourth possibility exists, namely, the possibility of the coincident existence of a cerebral tumour.

In certain cases the evidence obtained is sufficiently strong to enable one to estimate with a degree of certainty the origin of the hydrocephalus, as, for example, in the specific types and the post infective meningeal conditions.

A considerable proportion of cases, however, necessarily remain in which no definite idea can be formed of the etiology, and such a lack of knowledge is not after all serious, because an answer to the question is not essential in deciding on the line of treatment to be adopted. If possible, however, an attempt should be made to classify the case according to its etiology.

2 **The Type of the Hydrocephalus**—According to Dandy's classification the question would be put: Is the hydrocephalus obstructive or communicating in type? But we have explained our reasons for preferring to express the varieties as ventricular or extraventricular.

Recognizing that practically all cases of hydrocephalus are obstructive in type, it is necessary to demonstrate whether the obstruction exists somewhere in the ventricular system up to and including the point of exit of the fluid from the fourth ventricle, or whether it exists in the subarachnoid spaces (communicating). Three procedures are followed in demonstrating the answer to the question—

a **Lumbar Puncture**—Lumbar puncture may give a suggestive result, but not a definite answer to the question. An intraventricular hydrocephalus may show low tension in the spinal fluid, while an extraventricular hydrocephalus may show an increased tension, but variations exist and occasionally the sequelæ are reversed. Therefore, while this investigation may be suggestive, it cannot be accepted as absolute.

b **Intraventricular Injection of an Indicator, with Investigation of the Cerebrospinal Fluid**—Following Dandy's recommendation, we have used phenolsulphonephthalein as an indicator. It is important that the solution employed should be neutral, and we have employed the preparation supplied by Martindale. Inattention to the detail of the neutrality of the fluid may result in a sharp reaction characterized by temperature and signs of cerebral irritability.

The technique of injection is simple. One or other lateral ventricle is punctured with

the needle of a 'recoed' syringe, the syringe holding 1 c.c. of the phenolsulphonephthalein solution. When the ventricle has been entered 1 or 2 c.c. of cerebrospinal fluid are removed into the syringe barrel and allowed to mix with the indicator. The mixture is then injected into the ventricle, and the needle withdrawn. After an interval of time lumbar puncture is done, and the spinal fluid is allowed to pass into a test-tube containing a few drops of 25 per cent sodium hydrate solution.

Dandy recommends that thirty minutes should be allowed to elapse before the lumbar puncture is done, in our experience such a long interval is unnecessary. If the indicator can be recovered in the spinal fluid it will become obvious within five minutes and sometimes even less.

By the recovery of the indicator (as evidenced by the pink coloration of the cerebrospinal fluid when it comes into contact with the sodium hydrate) it may be accepted that the hydrocephalus which exists is not due to an intraventricular obstruction up to and including the point of exit of the fluid from the fourth ventricle. In other words, the recovery of the indicator means an extraventricular hydrocephalus (communicating type—Dandy), while the non-appearance of the indicator in the spinal fluid may be taken as demonstrating an intraventricular hydrocephalus (obstructive type—Dandy).

c The Recovery of the Ventricular Injection Substance from the Urine—It is said that normally the amount of fluid absorption which occurs in the ventricular system is negligible, it is estimated at less than 1 per cent in two hours. The bulk of absorption within the skull occurs in the subarachnoid spaces, from which area as much as 40 to 60 per cent of an introduced fluid is excreted after two hours' interval. Based on these considerations the recovery of phenolsulphonephthalein from the urine subsequent to its injection into the ventricles becomes a matter of importance, for if a ventricular hydrocephalus exists practically none of the indicator will be recoverable within a reasonable time (two hours), while if an extraventricular hydrocephalus is present, the indicator will be recoverable, though not to the same degree as in a normal case.

We have not put this method of investigation into routine use. It expresses the same knowledge as is gained from an examination of the spinal fluid, while its demonstration is a matter of greater difficulty. Therefore we have relied on answering the question regarding the type of hydrocephalus by the second method of investigation, the examination of the spinal fluid after the intraventricular injection of an indicator.

Investigation by these methods, and more especially by the second, carries the observer a step forward in so far as he is now able to classify the case of hydrocephalus into a ventricular or an extraventricular type. If it should happen that the case is an extraventricular one, we have not considered it necessary to proceed further with the routine examination.

By the injection of air into the spinal theca Dandy¹¹ has demonstrated after a ray examination the actual location of the adhesions which are responsible for this type of hydrocephalus. He has shown the arrest of the air at the base of the brain and its absence from the cerebral sulci. All these points are demonstrable in extraventricular hydrocephalus, but their demonstration does not aid us in the question of treatment, at least, with the methods at present available to us.

3 The Level of the Lesion—If, however, our original investigation has shown that the hydrocephalus is a ventricular one, further details of knowledge must be available before an intelligent operative treatment can be undertaken. The degree of hydrocephalus must be known, the situation of the obstruction must be localized. This knowledge is gained by the method of ventriculography.

The Technique of Ventriculography—Cerebral diagnosis and cerebral surgery would benefit if it were possible to introduce into the ventricular system a fluid opaque to the action of x-rays. Hitherto no fluid has been suggested which fulfils the necessity of opacity and yet does not irritate the delicate structure of the ependyma and choroid plexuses. The necessity of the non-irritating character of the fluid is all the more important when we consider that in intraventricular hydrocephalus the fluid may be isolated within the ventricles for a prolonged period of time—in fact, until it is artificially liberated or removed.

In the absence of a safe and yet efficient fluid, air has been employed, and in young children, in whom the ossification of the skull is not too advanced, it delineates with remarkable distinctness the ventricular outlines. The method is simple. One lateral ventricle is punctured, and to the needle an accurately fitting 'record' syringe of 20-c.c. capacity

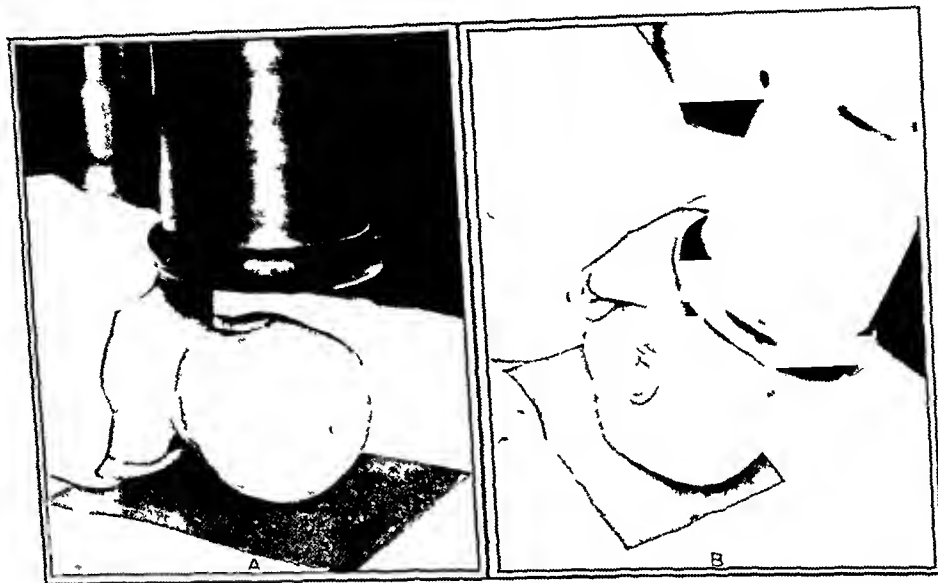


FIG 139.—The position of the head for ventriculography. A To record the outline of the left lateral ventricle. B To record the outlines of the third and fourth ventricles.

is fitted. The cerebrospinal fluid is slowly removed from the cavity of the ventricle, and when 20 c.c. are evacuated, a corresponding amount of air (20 c.c.) is injected into the ventricle, using the empty 'record' syringe for this purpose. The joint between needle and syringe is covered with sterile vaseline to prevent an escape of air at this point.

In a hydrocephalus of moderate size (18 to 19 inches in circumference) we remove 70 to 80 c.c. of fluid, replacing the fluid with a corresponding quantity of air. The removal of the fluid and the introduction of the air must be carried out in small alternating amounts, as the sudden removal of a large quantity of fluid without the supporting influence of the air might induce a cerebral oedema, and possibly an intraventricular hemorrhage. After the introduction of the air a hollow note can be elicited over the ventricle, and on movements of the head air may be heard to gurgle from one portion of the ventricular system into another.

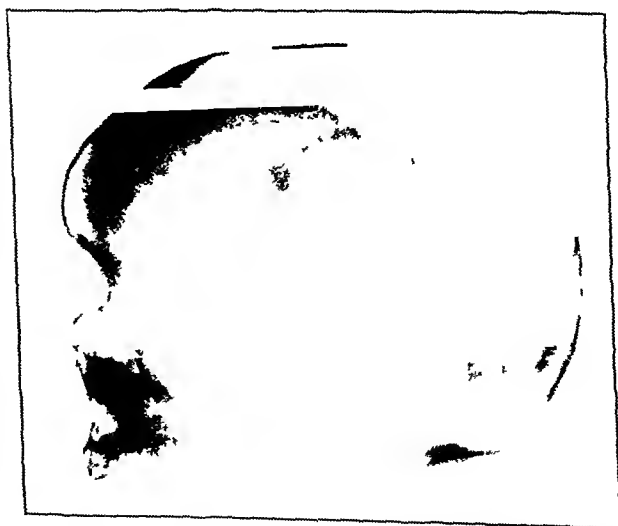


FIG 140.—Ventriculogram showing outline of dilated lateral ventricle filled with air. (Lateral position of head.)

Radiograms are now taken with the head in three different positions—right and left lateral, and with the head hanging downwards so that the skull base is at the highest level (Fig 139). The first plate will show the outlines of one lateral ventricle (Fig 140), the

second will demonstrate the opposite ventricle—assuming that the foramina of Monro and the third ventricle are patent—while the third plate will illustrate the third and fourth ventricles and the iter

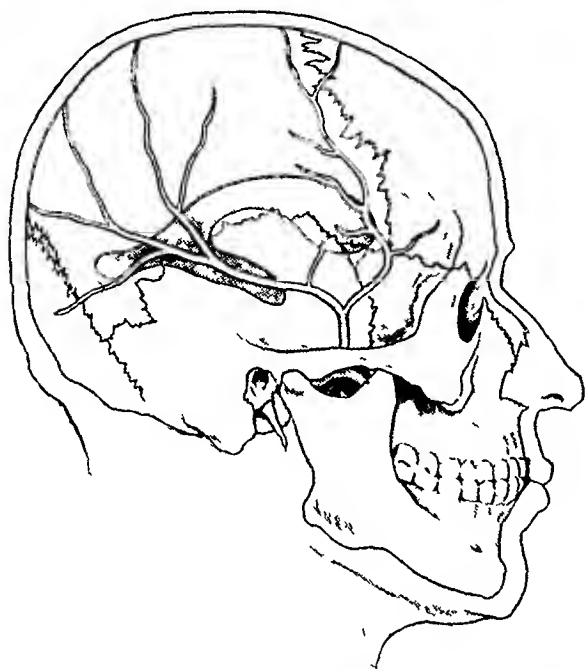


FIG 141.—Topography of normal lateral ventricle

Such views of the ventricular system would probably be very difficult to demonstrate in the normal brain (*Fig 141*). We have never made the attempt, but in the hydrocephalic, where the ventricular system is distended, definite and clear representations can be obtained. The value of the examination lies in the fact that it will locate the level at which the obstruction exists.

We have not had an opportunity of observing the appearance of an obstruction in the foramen of Monro, iter obstructions are characteristic (*Fig 142*), obstruction in the roof of the fourth ventricle is also distinctive (*Fig 143*), for, though ventriculography of an extraventricular hydrocephalus gives a somewhat similar representation, it can be otherwise excluded as shown above.

The introduction of air into the ventricles may appear to be a somewhat heroic proceeding in the investigation of a case of hydrocephalus

but actually the method is practically devoid of risk, certainly with the cases which



FIG 142.—Ventriculogram showing outlines of dilated left lateral and third ventricles. The air has failed to enter the aqueduct of Sylvius indicating an obstruction at this site—ventricular hydrocephalus of the third degree. (Inverted position of head)



FIG 143.—Ventriculogram showing outlines of dilated left lateral third and fourth ventricles. Note the air in the right foramen of Monro and the bulbar roof of the fourth ventricle. Obstruction at the roof of the fourth ventricle is indicated. Ventriculogram in conjunction with the coloured indicator test. (Inverted position of head)

have come under our care we have had no anxiety. Occasionally there is a reactive rise of temperature, but it is not excessive and it soon subsides.

A Survey of the Clinical Examination—A successful clinical examination will have thrown some light on three aspects of the disease (1) The possible origin, (2) The type—ventricular or extraventricular, (3) The degree of the ventricular type. With these facts at our disposal the consideration of the treatment may reasonably be discussed.

TREATMENT

The Importance of Early Treatment—If the efficient treatment of hydrocephalus is to give us satisfactory results it must be carried out before the expansion of the head has become too marked. There is nothing to be gained from operating on a child if the white matter of the brain is already reduced to a shell. No definite limit can be fixed; it must be left a good deal to the decision of personal judgement, but we have found that an increase in circumference measurement up to 3 inches above the normal is within the range of satisfactory post-operative recovery. Above this figure we enter the range of unsatisfactory results, and with every degree of increase the probability of improvement diminishes.

Various Lines of Treatment hitherto Suggested—Kausch,¹² in the course of a most exhaustive article, details the various procedures which have hitherto been tried in the treatment of hydrocephalus. We append the following tabular account, there is nothing to be gained by a more detailed recitation of the methods, and those who are interested are referred to Kausch's contribution (*see also* Haynes¹³).

The following methods have been suggested or actually used in the treatment of hydrocephalus—

- | | |
|--|--|
| 1 Intermittent drainage | $\left\{ \begin{array}{l} \text{i Of lateral ventricle} \\ \text{ii Of the spinal canal} \\ \text{iii By puncture of the corpus callosum} \end{array} \right.$ |
| 2 Continuous drainage | |
| <i>A</i> Of the lateral ventricle | |
| <i>B</i> Of the spinal canal | $\left\{ \begin{array}{l} \text{i To surface} \\ \text{ii Into retroperitoneal tissues} \\ \text{iii Into peritoneal cavity} \end{array} \right.$ |
| <i>C</i> Of the subarachnoid space (fourth ventricle) | |
| <i>D</i> Of the cisterna magna into the cranial sinuses | |
| 3 Indirect treatment (carotid ligatures) | |
| 4 Other methods of treatment such as by a seton, injection of iodine, galvanopuncture, compression of head, and by drugs | |

With such a variety of methods of treatment available the conclusion may be come to that no single one has proved efficacious. Incomplete methods of investigation and imperfect knowledge of the etiological pathology have been responsible for this unsatisfactory state of affairs.

The Essential Difference in Treatment between the Ventricular and the Extraventricular Types—It is obvious that a very clear distinction must be drawn from the operative point of view between the ventricular and extraventricular varieties. Both are certainly obstructive in nature, but, while one (ventricular) is localized and accessible, the other (extraventricular) is diffuse, and practically inaccessible.

If the hydrocephalus is of the ventricular variety—that is to say, if there is an obstruction at some portion of the ventricular system proximal to and including the roof of the fourth ventricle—experience has shown that the only efficient way of treating the hydrocephalus is by removing the obstruction, and so opening up the normal passage for the circulation of the cerebrospinal fluid.

In the extraventricular variety the problem is more difficult, but we discuss later the possibilities of treatment of this variety.

THE OPERATIVE TREATMENT OF VENTRICULAR HYDROCEPHALUS

All examples of ventricular hydrocephalus, with the exception of the very rare first and second degrees, are operated on by the occipital route. We have not had an opportunity of treating a case of the first or second degree, and would therefore refer the reader to Dandy's observations on this point. The essential feature of the suboccipital operation is to expose that portion of the ventricular system which is most accessible, namely, the roof of the fourth ventricle, and through this region to deal with or remove the obstruction which exists.



FIG 114.—*The suboccipital operation for hydrocephalus* 1—The position of the head upon the head rest is shown. The incision is indicated by the dotted line. The self retaining retractor has been fixed in place by a rubber band round the head. The anæsthetic is being administered by the intrapharyngeal method.

Preliminaries and Anæsthesia

—The operation should be undertaken while the child is in as fit a condition as possible. Nothing should be attempted if there is any suspicion of cold or bronchitis. Throughout the operation, which may be a lengthy one, the child is kept warm—an arrangement like a sleeping bag made of gamgee tissue meets the requirement. The child is placed on its face with the head flexed on the chest and, to ensure a satisfactory position, we have adopted a special head-rest which supports the head in the correct position and permits the administration of the anæsthetic and yet does not interfere with the respiratory movements (Fig 114).

Intrapharyngeal ether has been the anæsthetic of choice, the anæsthetic being administered by a ethereter introduced through the nostril.

The Exposure of the Roof of the Fourth Ventricle—A mid-line incision is made, extending from the external occipital protuberance downwards to the level of the 7th cervical spine. In the earlier cases we employed a crossbow incision, but it involved an unnecessary amount of hemorrhage, and the access which it provides is not appreciably greater than

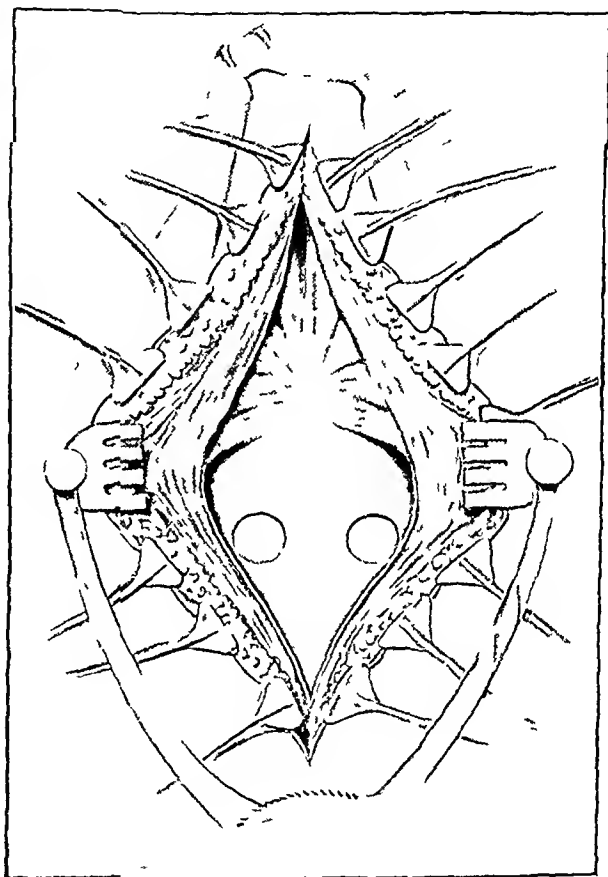


FIG 115.—*The suboccipital operation* 2—The vertical muscles of the suboccipital region have been split in the middle line down to the bone. The foramen has been detached from the occipital bone. These structures are retracted. The spine of the axis with the attachments of the deep muscles is exposed. The bone has been perforated with the burr over either cerebellar hemisphere.

that afforded by the straight mid-line incision. The incision is deepened through the fibrous intermuscular space, exposing the mid-line of the occipital bone and the spines of the upper cervical vertebræ (*Fig 145*). The muscular attachments are separated outwards, and such free separation is possible that we have not found it necessary to carry out any transverse division of the muscles. We believe that any transverse section of the muscles is to be avoided, the upper portions of the muscles in the child are flimsy and they are easily so bruised and destroyed as to make their re-attachment difficult. From the occipital bone the separation may be carried out subperiosteally, the trapezius need not be disturbed, but the complexus, the rectus capitis posterior minor, the superior oblique, and the rectus capitis posterior major are separated outwards to the line of the occipital artery, this vessel is not in view, as it is under cover of the separated muscles. The result is an exposure of the occipital bone, vertically from the 'linea suprema' to the margin of the foramen magnum, and laterally from the right to the left occipital artery line.

Two trephine openings are now made, one on each side of the mid-line, so as to avoid the occipital sinus. The trephine openings are made with a Hudson's drill, and enlarged with a burr. With a rongeur forceps a crescentic area of bone is removed, the concavity of the crescent being at the posterior margin of the foramen magnum, while the convexity is half an inch within the area of the muscular separation. The removal of the posterior margin of the foramen magnum is an important point. During this stage of the proceedings bleeding is troublesome, more especially at either side of the foramen magnum. It is arrested by the use of bone wax and by gauze plugs. Special care is required as the area of bone over the occipital sinus is removed, and the downward removal of bone should not be carried so far as to damage the emissary veins which pierce the bone a quarter of an inch from the lateral angles of the foramen magnum. The upward removal of bone need not expose the lateral sinuses.

The dura of the cerebellar fossa appears in view, and an estimation can be formed of the degree of intracranial pressure which exists by the amount of dural bulging which is present. If there is considerable tension we now puncture one lateral ventricle and remove a sufficiency of fluid to relieve appreciably the pressure which exists.

The next stage is the control of hæmorrhage from the occipital sinus and from the marginal sinuses. This is efficiently done by including the various sinuses in sutures carried on round needles. Four ligatures are applied, one at each extremity of the occipital sinus and one around each marginal sinus as far forward as the ligature can conveniently be placed (*Fig 146*).

The dura is now opened over each cerebellar hemisphere and it is carefully separated inwards until the folia cerebelli is reached. This structure is divided about its centre with a fine pair of blunt pointed scissors and immediately there is a retraction of the divided dural tissue and an exposure of the posterior surface of the cerebellar hemisphere covered by the piamenoid tissues. Additional space can be obtained, when necessary, by

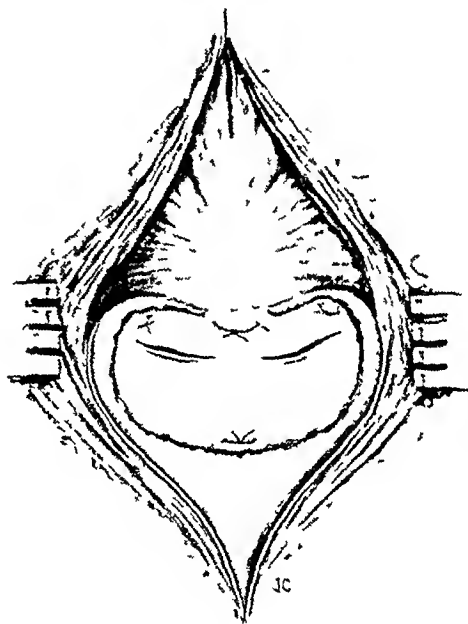


FIG 146.—The suboccipital operation. 2.—The bone defect has been completed and the posterior margin of the foramen magnum removed. Ligatures have been applied to control the occipital and marginal sinuses. The dura mater has been incised on either side of the mid line.

extending short vertical incisions upwards from either extremity of the transverse incision so as to form an upper dural flap. A special retractor, having a cup shaped surface for

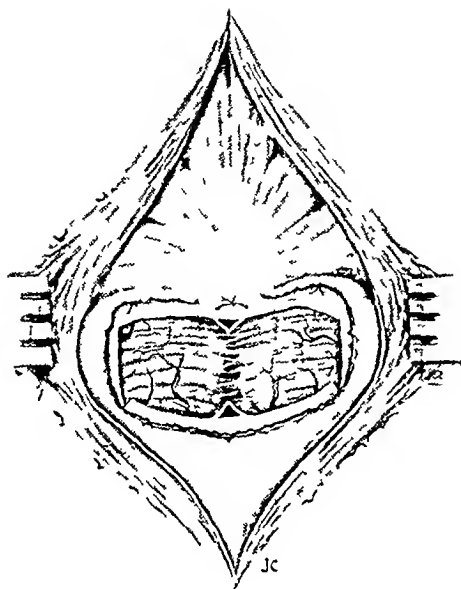


FIG. 147.—*The suboccipital operation.* 4.—The dural retractor has been completely opened and the upper flap allowed to retract. The cerebellar hemispheres and cisterna magna are exposed.

each cerebellar hemisphere and a central notch which is occupied by a small electric lamp, is now introduced beneath the cerebellum, and that organ is gently displaced upwards. The displacement must of course be regulated with the greatest delicacy and care. A space is exposed which lies between the under surface of the cerebellum and the posterior surface of the medulla, and which is bridged across by strands of arachnoid tissue, this is the cisterna magna (Fig. 147). If the hydrocephalus is of the fourth degree, the obstruction having occurred in the roof of the fourth ventricle, and if it is of an adhesive type, the result of a birth hemorrhage or a former basal meningitis, this space may be largely obliterated by fibrous adhesions and hemorrhagic extravasations. If such is the case, great care must be exercised in the separation of the adhesions, because, owing to the distention of the ventricles, the roof of the fourth ventricle is displaced backwards, and is torn before one is aware of its proximity. The roof of the ventricle should now come into view. If it is a bulging tent-like structure with definitely thickened texture and obvious obliteration of its foramina, it may be accepted that it is the site of the obstruction of the circulation of the cerebrospinal fluid, and be dealt with accordingly. It is our practice in such cases to remove a diamond-shaped area of the tela choroidea with fine spring forceps and eye scissors fitted with long-angled handles (Fig. 148).

The Relief of an Iter Obstruction.—If it should happen that the hydrocephalus is of the third degree, the obstruction existing in the iter, the operation has to be carried a stage further. In the third degree the fourth ventricle is not distended, and the obstruction in the iter is generally at one or other extremity. To gain access to the iter we follow the technique which Dandy first recommended. A small nasal speculum is inserted beneath the inferior vermis of the cerebellum, and, if further access is necessary, the vermis is split in the mid-line with a fine-bladed knife. The edge of the inferior medullary velum comes into view, and,

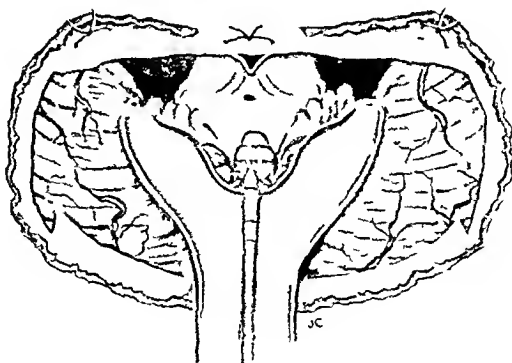


FIG. 148.—*The suboccipital operation.* 5.—The cerebellar retractor has been introduced and the cerebellum displaced upwards and its lobes laterally. The roof of the fourth ventricle is in view, illuminated by the small lamp. The foramina of Magendie and Luschka are seen (if not obliterated by adhesions).

if the tela choroides forming the lower half of the ventricular roof has not been opened up it is now perforated. A fine rubber catheter is guided into the upper angle of the ventricle, and is gently pushed onwards. The obstruction may be of such a fragile nature that there is little real resistance, and the catheter passes into the dilated third ventricle. This is signalized by a gush of cerebrospinal fluid. If the obstruction is more definite and cannot be overcome by the catheter, a millimetric probe is used to open up the closed communication (Fig 149), this instrument, however, must be used with extreme care, as any forcible manoeuvre may very easily result in perforation of the brain tissue around.

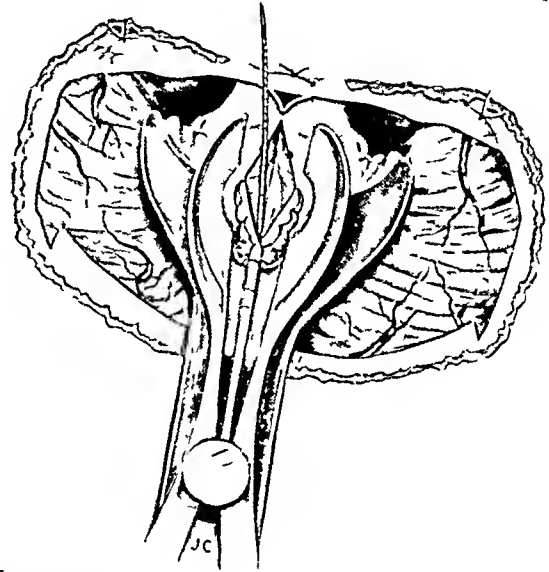


FIG 149—The suboccipital operation. C—Final step to relieve an obstruction in the aqueduct. The roof of the fourth ventricle has been opened and the lower part of the vermis split. With the aid of the nasal speculum the upper terminus of the fourth ventricle is exposed. A graduated probe is inserted through the aqueduct into the third ventricle.

After the channel has been opened with the probe the small rubber catheter is inserted, and it is demonstrated by the free escape of cerebrospinal fluid that the communication has been established.

As regards the further procedure, Dandy recommends that the rubber catheter should be left *in situ* for several weeks, the lower end, cut short being coiled up in the cisterna magna. He believes that this permits the formation of an epithelial lining around the tube, and diminishes the possibility of secondary closure. At first we followed this advice, but in the later series of cases we have been satisfied with efficient canalization of the *iter*, and it would seem that the restoration of the flow of the cerebrospinal fluid is sufficient to keep the channel open.

The Closure of the Wound—If the operation is to be a successful one, the relief of the obstruction must be followed by the most careful closure of the wound. A leak of cerebrospinal fluid to the surface is unlikely to become spontaneously arrested, and it ends almost certainly in sepsis and death. An attempt should be made to bring the dural edges together with a continuous catgut suture. The relief of tension which has resulted generally makes this procedure possible. The muscles are then closed in two layers, and the advantage of the straight incision over the crossbow incision now becomes evident, because the closure can be accomplished so much more efficiently.

In the immediate post-operative period attention must be paid in case the intracranial tension increases to such an extent as to lead to a leakage of fluid. If the tension is becoming marked ventricular puncture should be practised, and if necessary repeated.

THE TREATMENT OF EXTRAVENTRICULAR HYDROCEPHALUS

In this type of the disease the problem is in some respects more difficult than in the cases of the ventricular variety. The obstruction is a widespread adhesive one at the base of the brain and it is therefore inaccessible to direct removal. It has to be borne in mind, however, that certain cases of the extraventricular variety show a spontaneous arrest of the disease, a possibility which can never be hoped for in the intraventricular variety. At the present time the attempts at treatment have been directed towards diminishing

the production of the cerebrospinal fluid to such an extent as to bring it within the power of absorption which exists, for of course a certain degree of absorption still continues. There are two methods which have been used in securing this diminished production of cerebrospinal fluid.

Dandy¹⁴ has advised the removal of the choroid plexus from within the lateral ventricle, and he has published successful results in several cases. We have hitherto been content with less drastic measures. Intraventricular plexectomy is an exceedingly grave operation to perform, and there is no physiological proof that removal of a limited amount of choroid plexus proportionately diminishes the production of the cerebrospinal fluid. Stiles suggested the possibility of diminishing the production of the fluid by ligation of the common carotids, and in 1898¹⁵ and again in 1912¹⁶ he reported favourable results from this operation. It is clear, of course, that it is only the extraventricular type which will benefit from this procedure, its employment in the ventricular variety will inevitably lead to disappointment, and this may to some extent explain the adverse criticism which the method has received.

We have treated all the examples of extraventricular hydrocephalus which have come under our care by ligation of the common carotids, and the results have been sufficiently promising to warrant the continuation of this method. The actual results are

communicated later. The technique of the operation requires no detailed description—the vessels are tied at the crossing of the omohyoid muscle at an interval of ten days.

We confess that it seems unreal that ligation of the common carotid vessels should result in a diminished production of cerebrospinal fluid, as, through the medium of the anterior choroidal vessels, they are responsible for only a portion of the blood-supply of the choroid plexus. Nevertheless, we have definite clinical evidence that, following the procedure of ligation, a certain proportion of cases of extraventricular hydrocephalus become arrested. The procedure is such a simple one that it seems worth a more extended trial in the special type of case for which it is suited.

But the methods of plexectomy and carotid ligation are at best unsatisfactory. No definite result can be guaranteed, because they do not deal with the pathological condition which is responsible for the hydrocephalus, the subarachnoid ob-

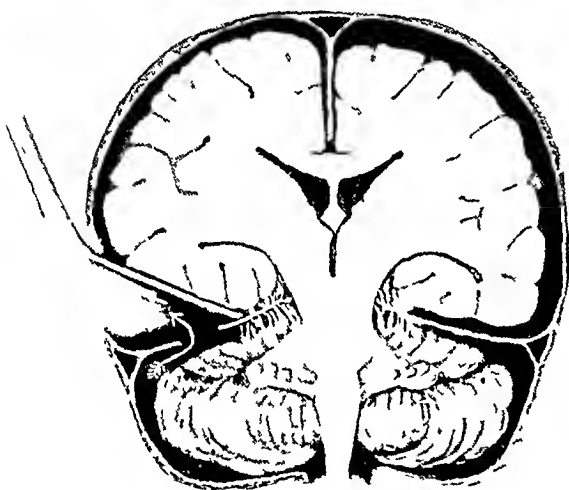


FIG. 150.—Suggested operation for the relief of certain types of extraventricular hydrocephalus. The pathway of the cerebrospinal fluid to the absorbing area of the cerebral subarachnoid space has been obstructed by adhesions between the free edge of the tentorium and the adjacent brain. By entering the skull above the lateral sinus and elevating the occipital lobe an opening has been cut in the tentorium outside the adherent area. The arrow indicates the new pathway for the cerebrospinal fluid.

struction. Considering the problem from the etiological point of view the obvious remedy is to open up a new pathway for the fluid in order to permit it to pass over the absorption area of the cerebral lobes. If the adhesions exist at the base of the brain, occluding the cisterns, it is difficult to see how any direct relief can be carried out, but if the adhesions occur between the free edge of the tentorium and the mid-brain, there are greater possibilities of operative interference. In the cadaver we have been able to carry out a procedure which we hope after further trial to employ in a certain variety of extraventricular hydrocephalus, the variety which is shown by ventriculography to be the result of adhesions between the mid-brain and the free edge of the tentorium. The proposed procedure is illustrated diagrammatically in the accompanying sketch (Fig. 150). The skull is trephined immediately above the lateral sinus, midway between the

mastoid process and the external occipital protuberance. The dura is opened, and the occipital lobe is elevated, the upper surface of the tentorium is exposed and this is carefully divided radially without damage to the lateral sinus. The incision through the tentorium is prolonged inwards as far as is safely possible. Through the incision thus made it is conceivable that a fresh channel may be established for the circulation of the fluid. The procedure is still hypothetical as far as its practical value is concerned, but it offers possibilities which we hope to test.

RESULTS

Tables 3 and 4 summarize the cases and the results obtained. Two cases of hydrocephalus associated with tumours are not included in this summary.

Table 3—VENTRICULAR TYPE

NO	NAME AND AGE	DEGREE	RESULT
1	J. S., 6½ months	4th	Died
2	F. A., 4½ months	3rd	Died
3	F. H., 9 weeks	4th	No operation
4	A. Y., 10 months	4th	Died
5	M. D., 5 months	3rd	No operation
6	R. M., 3 months	4th	Died
7	C. T., 7 months	4th	Cure
8	D. T., 7 months	4th (specific type)	Improved
9	N. S., 3 months	4th	No operation (died)
10	T. C., 1 year 4 months	3rd	Died
11	M. W., 4 months	4th	Arrested (probable cure)
12	M. C., 3 years	3rd	Improved
13	J. H., 6 months	3rd	Cure

Analysis of the Results of the Ventricular Type—At first sight the results appear to be exceedingly unpromising—out of 13 cases in 3 the condition was so advanced as to preclude any prospect of success from operative interference, 5 cases succumbed from the operation, in 3 cases the operative interference has resulted in apparent arrest of the disease, but we do not classify these as cures, because there has been no diminution in the bulk of the head, and the mental condition has remained permanently impaired, it is unlikely that these children will ever become useful members of society capable of taking their share in the work of the world. In 2 instances (and this is the hopeful side of the problem) it would appear that we have been successful in effecting a complete cure. The following is a brief epitome of the histories of these cases—

Case 1—C. T., 7 months. Female. Recommended by Dr. Brander, Fort William. On admission to hospital the child (Fig. 151) was suffering from a marked hydrocephalus; the fronto-occipital circumference was 20½ inches. The size and weight of the head was so great that it continually fell forward on the chest. Investigation showed that the hydrocephalus was ventricular and of the fourth degree. The child was operated on in May, 1921 and, as a matter of fact, it was one of the cases done during the afternoon demonstration at the Children's Hospital when the British Association of Surgeons met in Edinburgh. An obstruction was found in the roof of the fourth ventricle, and this was relieved. An excellent post-operative recovery was made.



FIG. 151.—Case of hydrocephalus, C. T. Appearance of child before operation. The hydrocephalus was a ventricular one of the fourth degree.

One year has now elapsed since the operation, and the accompanying photograph (Fig 152) bears witness to the child's present condition. The head has shrunk in size, the fontanelles are closed, and the sutures well ossified, the general intelligence of the child is unimpaired, it stands without assistance, and it is beginning to walk. The only evidence of defect which remains is an internal strabismus present in both eyes (Fig 153).



FIG 152—Case of hydrocephalus C F. Appearance of child one year after operation. An obstruction in the roof of the fourth ventricle was removed.



FIG 153—C F. Operated on for a ventricular hydrocephalus of the fourth degree when 7 months old. Present appearance of the child.

Case 2—J H, 6 months. Male (Dr Amslie, Edinburgh). The child, on admission to hospital, was found to be the victim of a ventricular hydrocephalus of the third degree. It was operated on in October, 1921, and the obstruction in the iter was relieved. A good recovery was made.



FIG 154—J H, age 12 months. Seven months after operation for a ventricular hydrocephalus due to an iter destruction.

Nearly seven months have now elapsed since the operation was performed, and a complete recovery appears to have been made. The head has diminished in size, and ossification is apparently complete except at the anterior fontanelle. The child is a sharp intelligent baby, and present appearances indicate normal development (Fig 154).

This proportion of cases (2 out of 13) is, of course, exceedingly small, but there are indications which promise better in the future. The operative procedure is of a highly technical character, and considerable experience is necessary if certain errors are to be avoided. Evidence of this is brought out by the fact that of the last 7 cases only 1 has succumbed after operation, 2 have been cured, 3 have been improved in so far as the condition has apparently been arrested, while one case was so extensive as to permit of no operative interference. We hope that in the future the knowledge we have gained may still further improve the results.

An Analysis of the Results of the Extraventricular Type.—In this group there were 6 cases only 5 were submitted to operative interference (ligature of carotids), one succumbing on admission to hospital. Of the remaining 5 cases, 1 succumbed within forty-eight hours of the first operation, as the result of hyperpyrexia, and 4 were successfully operated on. Of these 3 have very definitely improved, and we believe that the hydrocephalus is now

arrested, in the fourth case no improvement followed the operation, and the hydrocephalus increased to a fatal issue

Table 4—EXTRAVENTRICULAR TYPE

NAME AND AGE	METHOD ADOPTED	RESULT
P W, 4 months	Ligature of carotids	Condition arrested
S S, 8½ months	" "	Condition arrested
M M, 1 year 2 months	" "	Condition progressed (fatal in six months)
F S, 9 months	No operation	Successful on admission
N M, 9 months	Ligature of carotids	Condition arrested
A P, 10 months	Ligature of one carotid	Successful to hyperpyrexia within 24 hours of operation

Out of a total of 4 cases of extraventricular hydrocephalus with carotid ligature, 3 have very definitely benefited (Figs 155 and 156), and though we do not wish at this stage to classify them as complete cures we believe that everything points to a successful issue. The total series is, of course, too small to afford conclusive evidence but the proportion of 3 improvements out of 4 cases has so encouraged us that we intend to continue to practise this method of carotid ligature in cases of extraventricular (communicating) hydrocephalus in preference to the method of plexectomy.



FIG 155—S S, 8 months. An example of an extraventricular hydrocephalus. Before operation.



FIG 156—S S. Extraventricular hydrocephalus. The same child as shown in Fig 155, six months after ligature of carotids.

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A CLINICAL STUDY OF PHRENIC SHOULDER-PAIN, WITH SPECIAL BEARING ON THE DIAGNOSIS OF ACUTE ABDOMINAL DISEASE

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THE purpose of this study is to show the great importance, in the diagnosis of acute abdominal disease, of pain referred to the shoulder from the diaphragm and the adjacent parts supplied by the phrenic nerve. The occurrence of pain on the top of the shoulder in disease of the chest and upper part of the abdomen has been known for many years. Early knowledge on the subject has been well summarized by Kidd¹. A rational explanation of the symptom was first given in 1890 by Ferguson,² who showed that the phrenic nerve contained many afferent fibres. A very full clinical account of the symptom was published by Oelcker³ in 1914. The writer's observations began before Oelcker's work was brought to his notice, and it is thought that the conclusions reached extend beyond those published by the Continental observer.

Definition—By phrenic shoulder-pain is indicated pain felt on the top of the shoulder in consequence of an irritation of the terminations of the phrenic nerve. For the purpose of this study, only the occurrence of the pain consequent on the irritation of phrenic fibres in or adjacent to the diaphragm will be considered.

The referred pain caused by such irritation is felt over the areas of skin supplied by the same spinal segments which give origin to the phrenic nerve. The sensory distribution areas of the third, fourth, and fifth cervical segments are the parts involved, though of these the fourth segment is by far the most important. Roughly speaking, the pain is felt within the areas supplied by the descending cutaneous branches of the third and fourth cervical nerves. Sometimes the pain will be described as shooting down the outer aspect of the arm in the distribution area of the fifth cervical segment.

It is very necessary to distinguish this pain from that infrascapular segmental pain commonly felt in gall stone disease and various gastric conditions. Such pain is often loosely and misleadingly referred to as pain in the shoulder region, but it has no connection with the phrenic pain we are considering.

We do not include in this account that pain which has been described by some authorities as being felt on pressure over the trunk of the phrenic nerve in the neck, for we are rather sceptical as to whether one can entirely exclude superficial tenderness in pressing over the phrenic nerve.

The illustrative cases mentioned in the text are very necessary to the argument, for each has been chosen to illustrate some particular point. Nature is constantly making experiments on human beings, and by noting carefully the conditions existing in any experiment one may draw conclusions which have the force of scientific trial.

The Nature of the Pain—Phrenic shoulder-pain varies considerably in intensity and quality. Usually it has the qualities of an ache, and is regarded by the patient (and often by the doctor) as a rheumatic pain. Sometimes it is very sharp and stabbing, or it may feel as if a nail were being driven in at the painful spot. Very frequently the discomfort occasioned may not be severe enough for the patient to complain, so that it is necessary to inquire about the pain in every case if one is to avoid missing the symptom. Some patients say that they have no pain, but that there is a sensation of stiffness in the affected part.

Diseases in which Phrenic Shoulder-pain may occur—Any condition which may cause irritation of the diaphragm or the serous coverings of the diaphragm, or the contiguous tissues supplied by the phrenic nerve, may be the cause of phrenic shoulder-pain. It is clear, therefore, that diseases of the liver, stomach, duodenum, pancreas, and spleen below the midriff, and the pleura and pericardium above that muscle, will most commonly cause the pain. Inflammatory disease of the lower abdomen will only demonstrate the symptom if the inflammatory process reaches to the diaphragm. Sudden intraperitoneal hæmorrhage may, by pressing up under the diaphragm, irritate it sufficiently to cause shoulder-pain.

I have known the pain of diagnostic value in the following conditions —

Liver abscess	Appendicitis
Perforated gastric and perforated duodenal ulcer	Ruptured ectopic gestation
Subphrenic abscess	Dilated stomach
Cholecystitis with adjacent peritonitis	Actinomycosis of the thoracodiaphragmatic junction
Perforation of the gall bladder	Diaphragmatic pleurisy
Splenic infarct	Basal pulmonary infarct
Spontaneous rupture of the spleen	Pericarditis
Acute pancreatitis	

Hepatic Abscess—Perhaps in this condition more than in any other it is general knowledge that pain at the tip of or on the top of the shoulder may occur. It was in connection with liver abscess that my interest was first aroused in the subject of phrenic shoulder-pain whilst abroad during the war, and after a careful study of the pathological data furnished in the valuable monograph of E. J. Waring,⁴ the conclusion was reached that shoulder-pain accompanying liver abscess was seldom or never felt unless the pus was near to or threatening to perforate the diaphragm. This conclusion has been confirmed by clinical experience, so that one may state with confidence that in any patient with symptoms of liver abscess who has phrenic shoulder-pain the abscess must be close to and irritating the diaphragm. It is unnecessary to give examples of this condition, but it is important to remember that the shoulder-pain may be the only symptom calling attention to such an abscess.

Perforated Gastric and Duodenal Ulcer—Phrenic shoulder-pain is a very important symptom in the case of a perforated ulcer. Though not invariably present it is sufficiently constant to make it a necessity for the careful surgeon to inquire concerning the symptom in every acute abdominal case. It is necessary to inquire, since the shoulder-pain is usually overshadowed by the abdominal pain, and no spontaneous complaint may be made. Sometimes the referred pain comes on simultaneously with the abdominal, but occasionally it is delayed in onset. In the first case of perforated ulcer in which I noted shoulder-pain, it was only when the patient lay down in bed that the pain drew forth complaint.

With the perforation of a duodenal or pyloric ulcer the pain is usually felt in the right suprascapular fossa or over the right acromion process. Less commonly the pain is felt just above the right clavicle. With an anterior perforation of the stomach the pain is referred to the region of the left clavicle or left acromion process. If the perforation be near the cardia, and the escaping contents irritate the median portion of the diaphragm, pain may be felt over both acromioclavicular regions. I have known a patient complain also of a sense of weakness in the upper arm in addition to the shoulder-pain. It is not unlikely that this may have indicated that the sensory fibres of some of the arm muscles were reflexly affected.

Subphrenic Abscess—Phrenic shoulder-pain is present at some time or other in the evolution of nearly every subphrenic abscess, though, curiously enough, I have been unable to find any record of this very helpful symptom in any of the classical articles on the subject. The pain is generally felt only in the stage of active formation of the abscess. When once the abscess is localized no complaint of shoulder-pain may be made. The position of the referred pain varies according to the position of the abscess (*see below*).

Gall-bladder Conditions—Any inflammatory condition spreading from the gall-bladder may cause phrenic shoulder-pain. Acute cholecystitis does not cause it until the inflammation has spread beyond the confines of the viscus. The passing of a stone down the biliary ducts is not accompanied by top-of-the-shoulder pain unless there is accompanying inflammation round the ducts. Rupture of an infected gall bladder may cause the significant pain. In gall-bladder conditions the pain is generally referred to the right supraspinous fossa, occasionally to the acromiodeltoid region.

Splenic Conditions—Slow enlargements of the spleen do not lead to diaphragmatic irritation and its characteristic pain, but the local peritonitis consequent on a splenic infarct, and the irritation caused by collecting blood-clot, are sufficient to produce the symptom.

Case 1—Phrenic shoulder pain consequent on spontaneous rupture of the spleen

On April 6, 1922, a young man was admitted to the Bolingbroke Hospital with the following history. He had returned from active service in India on account of malaria, for which he had been recently treated. At 3 a.m. on the day of admission he had awakened with acute left sided abdominal pain. Two hours later he felt pain all over the top of the left shoulder. On admission at 3 p.m., he was collapsed and anæmic, had a tender tumid abdomen, and there were signs of free fluid in the abdomen. The diagnosis of spontaneous rupture of the spleen (which had been brilliantly made by his private doctor) was considered certain in view of the shoulder pain. Operation revealed much free blood in the peritoneal cavity and a collection of clotted blood forming a cast of the under surface of the left dome of the diaphragm. Splenectomy was performed and an excellent recovery followed. The splenic rupture from which the bleeding had occurred was not large.

Case 2—Infarct of spleen

M. L., female, admitted to St Mary's Hospital, Aug. 26, 1920, was found to be suffering from ulcerative endocarditis. She complained of pain over the left acromioclavicular joint. At the autopsy, Sept. 8, 1920, there was found an infarct of the central zone of the spleen and a small infarct of the upper pole.

Acute Pancreatitis—I have not myself seen a sufficient number of cases of acute pancreatitis to be able to dogmatize or generalize on its symptomatology, but in rarer diseases it is permitted to collate experiences outside one's personal practice. On *a priori* grounds I had long thought that acute pancreatitis should, by irritation of the left crus of the diaphragm, lead to phrenic shoulder-pain, but until recently, the cases I questioned never gave an affirmative answer to the inquiries concerning the pain. Just recently a case I saw at St James's Hospital presented the symptom. She was a woman, age 46, upon whom Dr MacCormac operated successfully for a very acute pancreatitis. On questioning her she was very emphatic that at the onset of the agonizing abdominal pain she also had pain in the left supraspinous fossa, which was relieved when the abdominal pain was relieved.

For an even better illustration of shoulder-pain in acute pancreatitis, I am indebted to the kindness of Mr Tudor Edwards. I give the account in his own words.

Case 3—Acute pancreatitis

"The patient was a rather fat woman, age about 60 with a previous history of undefined gastric disturbance. She was somewhat distended, and had general abdominal tenderness more marked in the left epigastrium. She was complaining of severe abdominal pain, and especially pain about the left shoulder. To the best of my recollection the pain appeared to be localized over the left supraspinous fossa. A rather interesting fact struck me at the time, namely, pressure over the abdomen over the pancreas increased the shoulder pain. At operation she had the usual signs of acute pancreatitis, brown effusion, fat necrosis, and a large distended pancreatic swelling with gall stones. Cholecystostomy with drainage of the pancreas anteriorly was done, and eventually complete recovery took place. Incidentally, there was no post operative complaint of shoulder pain."

It is unnecessary to comment on Mr Tudor Edwards' excellent description.

Acute Appendicitis—It is seldom that phrenic shoulder-pain is a symptom in appendicitis, for the very good reason that it is infrequent for the infection to extend up to the diaphragm.

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With a long ascending appendix, however, or in cases where the cæcum and appendix are much higher than normal, the symptom is to be expected. I can only find references to two or three such cases in Continental literature, and I have myself only known of two cases in which such shoulder-pain occurred.

Case 4—Perforation of a retrocæcal appendix

R. M. was taken with acute abdominal pain, followed about fifteen hours later by severe pain over the right acromial region. A perforated retrocæcal appendix was found at operation, and there was a great amount of seropurulent fluid in the abdominal cavity. For three weeks after the operation slight pain continued to be felt in the right shoulder on deep inspiration. The initial pain in this case was described as that of a nail being driven into the acromioclavicular joint.

It is specially to be noted in this case that the pain was not felt on top of the shoulder until some hours after the abdominal pain. This was also exemplified in the second case, in which pain was felt over the right clavicle twelve hours after the onset of the abdominal pain. At operation an inflamed ascending appendix with spreading peritonitis was found, and some lymph was noted in front of the liver.

Ruptured Ectopic Gestation—The interesting fact has long been known that in some cases of ruptured ectopic gestation pain may be felt over the clavicle. If the question be put as a routine it will be found occasionally that pain is complained of in the supraspinous fossa and over the acromioclavicular joint and deltoid. This pain is clearly due to sudden irritation of the diaphragm by the blood which pours from the pregnant tube.

Case 5—Tubal mole

A R., age 30, who had not nussed a period but whose last monthly loss had been much under normal, was seized with acute abdominal pain and vomiting on Dec 23, 1920. At the same time she had pain over both clavicles. Her condition improved for a day, but was worse on Dec 25, when she fainted and had a rigor. On Jan 2, 1921, she was admitted to the Bolingbroke Hospital, where I removed a right tubal mole. There was much blood-clot in the pelvis.

The clavicular pain was only felt with the first severe hæmorrhage, when it may be surmised the blood flooded the anterior subdiaphragmatic region.

Actinomyces of the Thoracodiaphragmatic Junction—When actinomyces attacks the chest it is almost always at the base, and the diaphragm is usually attacked early. Though a slow inflammatory process, the condition may cause sufficient irritation to produce the characteristic shoulder-pain. The pain may seriously mislead, and on the right side may give rise to a mistaken diagnosis of gall-stones, as in the following instance.

Case 6—Actinomyces mistaken for gall stones

A man came under my care on Dec 1, 1913, for abdominal pain. He had been well until three weeks prior to that date. He had at that time been taken with very severe pain on top of the right shoulder and in the right hypochondrium. The pain had lasted, with occasional intermissions, for the three weeks. There was no jaundice, but some tenderness and rigidity in the right hypochondrium. He was admitted to hospital, where a physician colleague diagnosed duodenal ulcer or possibly subphrenic abscess. My own opinion, based on the shoulder-pain (the correct significance of which I did not at that time understand), inclined to gall stones. Operation revealed a rather large liver and distended gall-bladder, but no sign of gall stones or other disease. I drained the gall bladder. He was not much relieved. A month later he developed a cough, and a large fluctuating swelling was noted in the right loin. This was opened and from it were obtained characteristic actinomycotic granules. The diagnosis was confirmed microscopically.

Since this case I have had another patient with actinomyces involving the left thoracodiaphragmatic junction who had pain in the left supraspinous fossa, and the local and referred pain diminished and increased together.

It is not within the province of this study to discuss phrenic shoulder-pain of thoracic origin save in the section on localization-value of the pain.

The Localizing Value of Phrenic Shoulder-pain—Clinical evidence supports the view that the position of the referred pain on the top of the shoulder varies according to the part of the diaphragm irritated. This conclusion is at variance with that reached by Capps as the result of his experimental irritation of the diaphragm in cases of pleural effusion. He states that the maximum pain-point in the neck in a given individual was

the same from whatever part of the diaphragm it was elicited" It is possible that the different results may be due to the difference between a mechanical stimulus and that due to inflammatory conditions

It is necessary, therefore, to put on record the evidence which points to the view that there is a correspondence between the part of the diaphragm affected and the region of the shoulder over which pain is referred It is agreed by all observers that irritation of the right side of the diaphragm causes pain on the right shoulder, whilst left shoulder-pain results from some affection of the left portion of the diaphragm This is a general rule to which I have seen but one—and that a doubtful—exception The localization which has apparently been overlooked by other observers and which is at variance with Cripps' conclusion may be summarized as follows Irritation of the anterior part of the diaphragm causes pain in the corresponding clavicular or supraclavicular regions, irritation of the posterior part of the diaphragm causes pain in the supraspinous fossa of the same side, irritation of the top of the phrenic dome causes pain in the corresponding acromio-clavicular regions, and finally, pain felt over both shoulders indicates a median diaphragmatic irritation

Evidence that Irritation of the Anterior part of the Diaphragm causes Pain in the Anterior part of the corresponding Shoulder Region

Case 7—Subphrenic abscess

In September, 1920, a youth was admitted to the Bolingbroke Hospital with symptoms of general peritonitis He was so ill that the resident surgeon who operated thought it wise merely to drain the pelvis The general condition thereafter improved, and the fever abated Ten days later he complained of the occasional occurrence of pain exactly over and along the left clavicle The medical officer (Major Lowe) examined the left lung base, discovered signs of pleurisy, and asked me to see the patient Examination showed dullness, and diminished breath and vocal sounds at the left base posteriorly The presence of fluid did not account for the clavicular pain, but on listening to the front of the left chest at the level of the diaphragm pleuritic respiratory crepitations were easily detected The history of the case, combined with the irregular fever and clavicular pain, pointed to the presence of a subphrenic abscess in the left anterior region Sub-pleural resection of the anterior part of the left 10th rib was performed, and a subphrenic abscess, which occupied a situation just in front of the spleen, opened and drained

Case 8—Diaphragmatic pleurisy

In August, 1920, I saw a patient with acute pain in the right side of the abdomen suggesting an intra-abdominal lesion The illness began with an acute stabbing pain in the right sub-clavicular fossa Examination revealed nothing abnormal in the abdomen, but below the right clavicle was a hyperalgesic area, and on auscultation of the chest a soft sticky pleural crepitation could be heard at the lowest level of the right pleura in front No adventitious sound and no dullness could be found posteriorly The temperature was 103° A diagnosis of diaphragmatic pleurisy was confirmed by the after course of the disease Slight basic pneumonia developed, but a good recovery ensued

Case 9—Pulmonary embolism due to malignant disease

A woman with rapidly-growing malignant intra-abdominal growth was taken with sudden acute pain under the right breast accompanied by pain in front of the right shoulder and the lower anterior part of the neck The right arm also felt weak The attack was accompanied by breathlessness, and a pulse of 140 Both pains had disappeared when I saw her ten days after the attack There could be little doubt that the attack was due to a pulmonary embolus with anterior diaphragmatic irritation

Perhaps the most instructive case that I have met with showing the value of phrenic shoulder-pain is the following

Case 10—Perforated ulcer near the cardia, coupled with a subphrenic abscess

In 1920, a young woman was admitted to the Bolingbroke Hospital with a history that five hours previously she had awakened with terrible pain in her abdomen and severe pain in both shoulders When I saw her soon after admission she presented the signs and symptoms of a perforated gastric ulcer She stated that the pain on the shoulders was almost as severe as that in the abdomen Asked to localize the shoulder pain she pointed to the site of the acromio-clavicular joint on each side When I gently touched the spots indicated she cried out with pain I concluded the median part of the diaphragm was irritated by a perforated ulcer near the cardia, and this was confirmed in opening the abdomen

The ulcer-perforation was sewn up and when I saw her again six hours after the operation the shoulder pain had disappeared. Five days after the operation pain was again felt over the left acromioclavicular joint. Two days later there was a little fever, and on the tenth day after operation dullness was detected at the left lung-base, posteriorly. Concluding that there was a left subphrenic abscess I resected a part of the left 10th rib. Finding that the pleura came down almost to the costal margin, I made a second incision at the costal margin anteriorly, passed my finger up, lateral to and in front of the splenic flexure of the colon, and opened a large stinking left anterior subphrenic abscess. A large rubber drainage tube was inserted. After the opening of the abscess the peritoneal pain disappeared, but pain was complained of all along the left clavicle. Thus, when the irritation was removed from the left dome of the diaphragm and the anterior portion of the muscle was irritated by the rubber tube and pus, the pain was transferred to the clavicular region.

If this view of the localization of the referred pain be accepted, it provides a ready explanation for the symptom of pain over the clavicles noted in some cases of ruptured ectopic gestation, and mentioned by de Quervain in his excellent book. The blood poured into the peritoneal cavity most readily ascends in front of the intestine and would impinge upon and irritate the anterior part of the diaphragm. It is necessary to state, however, that in cases of extra-uterine pregnancy which rupture it is not uncommon for the patient to have pain in the supraspinous fossa of one or other side—a fact easily explained by the collection of clot against the posterior part of the diaphragm.

Evidence that Irritation of the Posterior part of the Diaphragm (and the adjacent parts supplied by the Phrenic) leads to Pain in the Suprascapular Region

In the first place liver abscesses, which in the majority of cases occupy the posterior and upper part of the right lobe of the liver, most commonly cause pain in the right suprascapular region. A fact so well known does not call for illustrative cases, but anyone who desires confirmation of the statement has but to study carefully the excellent pathological data of E. J. Waring. Secondly, in cases of perforated pyloric ulcer causing immediate irritation in the region of the right crus and the subhepatic area of the diaphragm, the pain is usually felt in the posterior part of the right shoulder region. Thirdly, in those few cases of pancreatitis which are accompanied by shoulder-pain, such pain is referred to the left suprascapular region.

It is sometimes possible in clinical work to obtain a direct stimulation of one particular region, thereby confirming or confuting an opinion, as in the following instance —

Case 11 — Phrenic Irritation caused by drainage tube

A patient under the care of a colleague underwent cholecystectomy, at the end of which operation a rubber tube was inserted down to the stump of the cystic duct. After the operation the patient complained of very severe pain in the right supraspinous fossa. The stitch fastening the tube to the skin was released and the tube withdrawn about an eighth of an inch. The suprascapular pain immediately ceased.

By consultation with anatomists I have carefully attempted to ascertain exactly where the undue pressure would have been made by a tube in the position indicated above, and the conclusion is that it was pressing against the peritoneum over the vena cava and the right crus at the posterior boundary of the foramen of Winslow. That this is no isolated case may be judged by the following case related to me by Mr. Eric Pearce Gould —

Case 12 — Another case of irritation by drainage tube

Miss L. Operation Feb. 7, 1920. Cholecystectomy was performed. When seen one hour after, she was complaining of severe pain of a gripping character in the right supraspinous fossa. Her binder was loosened and she dozed, on waking the pain was gone and she had forgotten all about it.

Here again the lessening of pressure causes the cessation of the pain, and in each case the pressure of the tube against the right crus and vena cava caused pain in the corresponding supraspinous fossa.

The above case illustrations afford strong presumptive evidence, if not proof, that there is a correspondence between the part of the diaphragm irritated and the position in which the referred pain is felt on the shoulder.

Is there any Means of Distinguishing the Phrenic Shoulder-pain caused by Thoracic Disease from that caused by Abdominal Disease?

It may be stated at once that there is no certain way of distinguishing the source of the pain, but it is sometimes possible to gain assistance of value by noting the exact position of the pain.

There can be little doubt that acute pleural pain is due to friction between the opposing pleural surfaces. Diaphragmatic friction is naturally most common in the region of the costodiaphragmatic sulcus. In a person who is the subject of acute disease the recumbent position is usually assumed, and any pleural effusion gravitates to the posterior costodiaphragmatic sulcus. Friction is therefore not so likely to persist in the posterior sulcus. In cases with slight effusion, friction in the anterior costodiaphragmatic sulcus may not be interfered with by the fluid poured out. It is likely therefore that in a series of cases of diaphragmatic pleurisy there will be a larger proportion of patients who have pain referred from the extreme anterior part of the diaphragm. From this region pain is referred to the corresponding clavicular and subclavicular regions. It is a clinical fact that acute subclavicular pain of phrenic origin is more commonly of thoracic than of abdominal origin. On the right side such subclavicular pain is usually of thoracic origin (*see Case 8*). On the left side subclavicular pain may also be caused by perforation of an ulcer on the anterior wall of the stomach near the cardia.

When the pericardial portion of the diaphragm is affected the pain is either just above, over, or below the left clavicle.

Case 13.—Phrenic shoulder-pain due to pericarditis

W. M., age 20, was admitted to St. Mary's Hospital on March 19, 1921. It was elicited that on March 8 he had been taken with a sudden acute pain under the left collar-bone. The pain lasted half an hour. It came on again when he lay down at night, and seemed to catch him when he took a deep breath. It got worse until March 19, when the pain was continuous and did not alter with the position of the patient. There was also pain in the epigastrium and along the right costal margin. On admission he was cyanosed. On March 21, a faint pericardial rub was heard just outside the sternum in the 3rd left intercostal space. A few days after admission he still had a little pain in the left subclavicular region, and the left arm felt a little numb over the deltoid.

Phrenic Shoulder-pain in Gall-bladder Disease.—It has long been an accepted teaching that in biliary colic the pain may radiate to the top of the right shoulder. This dogma has been transmitted from one generation to another and from text-book to text-book without any discrimination between the varieties of biliary colic, and for the most part without any operative or post mortem account of the condition which caused the referred pain. Observation of a consecutive series of abdominal cases with pain on the top of the right shoulder will easily demonstrate that—

1 Gall stones and gall-bladder disease are less commonly the cause of phrenic shoulder-pain than are perforated pyloric or duodenal ulcer.

2 Neither cholecystitis nor impaction of a stone in the cystic duct causes pain on the top of the right shoulder unless there is accompanying local peritonitis.

3 A stone impacted in the common duct does not cause pain on the top of the shoulder until congestion and œdema of the adjacent parts result.

The first of these three points might not hold good in the case of any particular surgeon, but in the general series of cases which fall to the lot of the ordinary surgeon it is certainly true.

Regarding the second point, I put it on record that I have never yet seen a case of gall-stones exhibiting pain on the top of the right shoulder unless there has been accompanying local peritonitis, or congestion and œdema of the neighbouring parts.

Some confusion has arisen because many observers loosely and incorrectly speak of "pain in the shoulder" when they mean pain under the right scapula. Before any comparable data can be obtained it is necessary for the observer to state clearly and exactly where the pain was felt and not to record merely "pain radiating to the shoulder or shoulder-blade."

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I append representative cases of gall-bladder disease to show the kind of pathological change which does or does not give rise to the shoulder-pain

Case 14—Stone in the cystic duct—no shoulder pain

Mrs C, admitted to the Bolingbroke Hospital in October, 1920, with pain in the right hypochondrium and right infrascapular region. No pain on the top of shoulder. No abdominal rigidity. Tender gall bladder easily palpated. At operation, a very distended gall bladder filled with thick mucoid fluid was found, and a stone discovered blocking the cystic duct.

Here there was no local peritonitis and no phrenic pain.

Case 15—Gall-stones, acute cholecystitis, and commencing peritonitis, with pain on top of the shoulder

Mrs C, age 68, who had had three attacks of pain in the right hypochondrium during the last few years, was taken ill five days before I saw her, in April, 1920, with pain in the right upper abdomen radiating to the right infrascapular region. When I saw her the temperature was 100° and, though there was no abdominal-wall rigidity, there was pain on pressing deeply over the gall bladder. No pain had been felt at the top of the shoulder. She was moved to hospital in an ambulance, and the first thing she said to me when I saw her there was that on the journey she had felt pain all over the top of the right shoulder. Operation showed acute cholecystitis, omental adhesions, free fluid, and spreading peritonitis; one large gall-stone was found in the gall bladder.

In this case my inference was that the examination and removal of the patient caused the extension of the inflammation to the tissues covering the diaphragm.

Case 16—Stone in the common duct. Pain in the top of the shoulder starting when the abdominal pain ceased

Mrs M T, age 35, admitted to the Bolingbroke Hospital in February, 1922, with a history of twenty-four hours' acute abdominal pain followed by jaundice. No pain in the back. When the jaundice began and the abdominal pain ceased she began to feel pain at the back of the neck (in the suprascapular fossa at the anterior border of the trapezius). Jaundice gradually became worse. On Feb 24, I operated and removed a gall-stone from the supraduodenal part of the common bile-duct. The gall bladder was full of stones, which were removed. This patient was quite certain that the shoulder-pain (which she thought was rheumatic) did not begin till the abdominal pain ceased and jaundice began. At the operation, the tissues round the foramen of Winslow were oedematous and inflamed, though the gall bladder itself was not very inflamed.

In this case no phrenic shoulder-pain was felt whilst the abdominal pain, caused by the stone passing through the cystic duct, persisted, but so soon as it stuck in the common duct oedema and inflammatory reaction followed, and irritation of the neighbouring phrenic nerve endings at the posterior margin of the foramen of Winslow caused the shoulder pain.

In the days when the abdomen was not opened for gall-stones while the patient was alive, there was excuse for the view that pain on the top of the shoulder was caused by the passing of a stone. A phrenic twig to the liver or biliary ducts served as sufficient anatomical reason for the fact of such pain. In *Reynolds' System* it was stated that in biliary colic the pain sometimes radiated to the clavicles. In *Bristowe's System* it was asserted that the pain might radiate to the shoulder-tip. One of the best modern *Systems* is more indefinite in stating that "pain radiates over the abdomen and to the right scapula, but in some cases the pain radiates to the left shoulder." Biliary colic usually causes pain just below the inferior angle of the right scapula, but seldom causes it in the acromioclavicular region. In considering biliary colic and referred pain caused thereby, one must realize that pain similar to that caused by the passing of a stone can be caused by inflammatory conditions and that the passing of a gall-stone along the ducts may in itself lead to contiguous inflammatory changes.

Kehr states that 'one thing can be confidently asserted. It is by no means necessary (for biliary colic) that a stone should stop up the duct, for an inflammation of the gall bladder without stone, and a swelling of its ducts, can produce a pain indistinguishable from biliary stone colic. He continues, 'It would in my opinion be better if we abandoned the term stone colic for the stone as a foreign body causes generally no pain. I have found a stone stuck in the neck of the gall-bladder and in the duodenal papilla

without the patient having any pain. Why? Since inflammation was absent in each case."

In *Osler and McCrae's System of Medicine* an enlightened view is expressed: "In the great majority if not in all cases local examination (in gall-stone colic) reveals tenderness, abdominal rigidity, and the other local phenomena of acute cholecystitis." It is also stated that "gall-stones are absent in 15 per cent of patients who have colicky pains." The writer concludes that "gall-stone colic should be interpreted rather as evidence of acute cholecystitis or acute exacerbation of chronic cholecystitis."

Some years ago Mr Mayo Robson⁷ wrote an interesting note on three cases in which pain at the tip of the shoulder was a prominent symptom, and in none of which was it caused by gall-stones. All three were due to tumours growing at the upper end of the kidney. The explanation offered for the pain was that a small branch of the phrenic passes to the semilunar ganglion. In view of the necessary irritation of the tissues over the diaphragm by such tumours, that explanation is hardly needed.

The Value of Phrenic Shoulder-pain in Diagnosis and Differential Diagnosis—It should be a routine question to ask every patient suffering from acute abdominal pain whether pain is also felt on the top of one or both shoulders. The exact site is best indicated by placing the hand gently over the acromioclavicular region whilst the question is put. There can then be no ambiguity as to the part meant by 'the shoulder'.

The absence of shoulder pain is perhaps not of so much importance, but the presence of suprascapular, supra-acromial, or supra- or subclavicular pain is always of great value in diagnosis, as indicating an irritation of the diaphragm or its serous coverings.

Such pain is often a distinguishing point in differential diagnosis. The conditions between which it may help to distinguish may usefully be enumerated.

1 *Differential Diagnosis of Acute Appendicitis and Perforated Duodenal Ulcer*—With a perforated duodenal ulcer it is the rule for the patient to feel pain in the right suprascapular fossa (or over the right acromioclavicular joint and deltoid) simultaneously with or soon after the pain in the abdomen. Rarely does the shoulder-pain delay its onset for a few hours. In appendicitis, on the other hand, pain on the top of the shoulder is very rare, and in the rare cases of its occurrence the pain is not felt for some hours after the onset of the pain in the abdomen. If phrenic shoulder-pain develops some days after an acute attack of appendicitis, one must examine carefully for subphrenic abscess.

2 *Diagnosis between Perforated Gastric and Perforated Duodenal Ulcer*—With a perforated pyloric or duodenal ulcer, the shoulder-pain is usually felt over the right suprascapular fossa, the right acromioclavicular joint, and deltoid, whilst a perforation of the anterior wall of the stomach causes either pain over both acromioclavicular regions (median irritation of diaphragm), or pain over or under the left clavicle (irritation of anterior part of left dome of diaphragm).

Case 17—Perforation of the anterior wall of the stomach

David B., age 49, admitted to St Mary's Hospital, Nov 19, 1921. He had been taken with acute abdominal pain at 9.30 a.m. the same day, and had also experienced slight pain under the left clavicle. There had been no pain in the right shoulder. General abdominal rigidity was present. The liver dullness was normal in the right axilla, but slightly diminished in front. From the presence of subclavicular pain I diagnosed a perforation of the anterior wall of the stomach, well to the left of the middle line. My colleague, Mr Clayton Greene, operated, and found a perforation in the position forecasted.

3 *Perforating Ulcer and Acute Pancreatitis*—If the arguments as to the localizing value of phrenic shoulder-pain be accepted, it should follow that pain in the left suprascapular fossa accompanied by general abdominal symptoms of severity would most likely be due to acute pancreatitis. A posterior perforation of the stomach might cause such pain but such a lesion would more likely cause adhesions and not be so acute in its onset. Any patient suffering from very acute abdominal symptoms in whom thoracic lesions can be excluded, may be reasonably supposed to be the subject of acute pancreatitis if pain

is simultaneously experienced in the left supraspinous fossa. It must be admitted, however, that this diagnostic help is only present in a minority of the cases.

4 *Right-sided Pleurisy and Abdominal Conditions*—The right anterior part of the diaphragm is relatively seldom irritated by acute abdominal lesions, but pleurisy frequently attacks the corresponding superior surface of the muscle at the anterior costophrenic sulcus. If, therefore, a patient complains of abdominal pain and pain at the same time in the right subclavicular fossa, the most likely pathological lesion is right-sided diaphragmatic pleurisy.

5 *Ectopic Pregnancy and Perforated Gastric Ulcer*—Sudden hypogastric pain associated with symptoms of collapse and accompanied by shoulder-pain in an adult woman should make one think of ectopic gestation. If the pain is felt over both clavicles or over the right clavicle, the diagnosis of ruptured ectopic pregnancy is almost certain.

CONCLUSIONS

Pain of a referred nature is frequently felt on the top of the shoulder as a consequence of stimulation of the sensory terminals of the phrenic nerve in or near the diaphragm. The pain is felt in some part of the segmental areas corresponding to the 3rd and 4th, and sometimes even the 5th cervical segments of the spinal cord.

This referred pain is met with in many conditions which cause inflammation or irritation of the diaphragm or contiguous structures.

There is an important localizing correspondence between the part of the diaphragm irritated and the position of the referred pain on the shoulder.

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ON SOLITARY FIBROMYXOMATA OF PERIPHERAL NERVE-TRUNKS, WITH A DESCRIPTION OF A CASE OF CYSTIC FIBROMYXOMA OF THE MEDIAN NERVE

By ERIC A. LINELL, MANCHESTER

M. G., a woman, age 42, came to the out-patient department of the Ancoats Hospital in October, 1921, complaining of a lump in the right arm, which she had noticed growing gradually for four years, and more rapidly during the last month. When first noticed she stated it was the size of a hazel nut.

Symptoms—Patient's only complaint was that occasionally she had attacks of shooting pain from the site of the tumour down into the middle finger. She suffered no disability whatever, and had noticed no impairment either of muscular power or of sensation in the limb.

Examination—This revealed a well-defined, painless, fusiform swelling on the antero-internal aspect of the upper arm slightly above the internal condyle of the humerus. The tumour was the size of a pigeon's egg, freely movable laterally, but not in the long axis of the limb. It was unattached to skin, and elastic to the touch, but fluctuation was not demonstrated. The essential connection of the tumour with the median nerve was not considered on account of the complete absence of motor or sensory symptoms. From the proximity of the tumour to the line of the nerve it seemed reasonable to ascribe the shooting pains in the median area to pressure, and a diagnosis was made of a soft fibroma arising from the deep fascia.

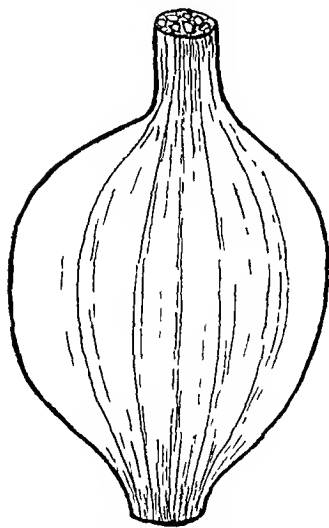


FIG 157.—Naked eye appearance of cyst after removal

Operation—This was performed on Nov. 21, 1921. Exposure of the tumour revealed a dark-blue fusiform swelling which appeared to interrupt completely the continuity of the median nerve. The swelling was definitely cystic, and incision allowed the escape of a considerable quantity of dark, fluid blood. As from naked eye inspection it appeared impossible that there should be any nerve bundles connecting the proximal and distal portions of the trunk along the cyst wall, some doubt was thrown on the accuracy of the preliminary clinical examination. After careful consideration and in view of potential malignancy, the median nerve was resected half an inch above and half an inch below the tumour. This was removed, and primary end-to-end suture of the resected

ends of the nerve was easily performed, assisted by flexion of the elbow. The tumour showed no adherence to surrounding tissues, and both resected ends appeared quite healthy. The skin incision was closed and the limb bandaged in full flexion at the elbow. Healing took place by first intention, and the patient was discharged from hospital on Dec. 8 with, of course, a typical median nerve paralysis.

A sketch of the specimen as removed at operation is reproduced in Fig. 157. After the specimen had been hardened in formalin, nerve-fibres were seen to be spread out in the wall of the cyst as shown in the sketch.

Subsequent Course—The patient has been having massage and electrotherapeutic treatment since the operation. Professor Stopford examined her on March 17 1922 nearly four months after the operation, and he considers that there are early signs of regeneration in view of a slight diminution in the area of lost protopathic sensibility and a positive Tinel's sign. There is no evidence yet of any motor recovery.

Histology—In view of the extreme rarity of cystic tumours of nerves, it seemed worth while to make a detailed histological study of the specimen. The complete absence of

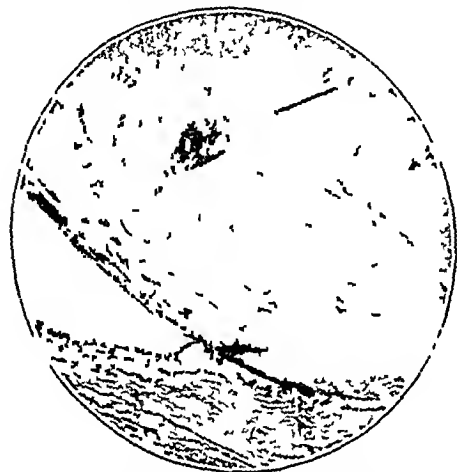


FIG 158—Section of cyst wall, low power magnification shows fibrous tissue with myxomatous areas. Note well formed blood vessels.

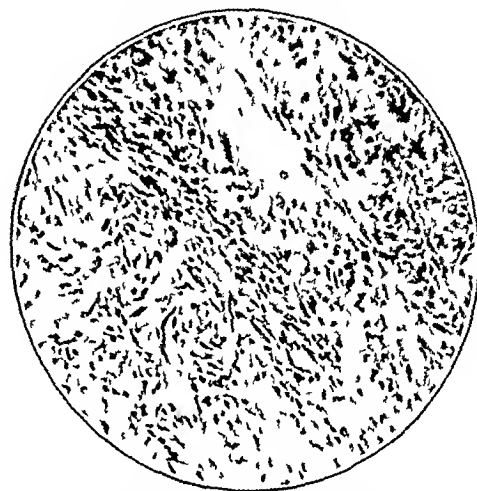


FIG 159—Wall of cyst, high power magnification shows the two main tissue elements of tumour.

motor and sensory symptoms in the case of a nerve tumour, which macroscopically gave no clear indication as to its nature, justified the histological investigation of the nerve above and below the lesion, both for signs of extension of the growth and of nerve-degeneration below the tumour.

Transverse sections of (1) the cyst wall, (2) the nerve above, and (3) the nerve below the tumour, have been stained with hemalum and eosin and in addition, transverse sections distal to the tumour have been stained by Weigert's method to show the condition of nerve-fibres distal to the lesion.

1. *The tumour*, as seen in a transverse section of the cyst wall, consists for the greater part of a very loose reticulum of branching cells with well marked nuclei. Mitotic figures are not seen and the blood-vessels throughout the section are well formed. Dr Charles Powell White considers that these cells bear a strong resemblance to the essential cell element of a glioma but as a glioma of a peripheral nerve is unknown he thinks it more probable that they are young fibroma cells. Scattered throughout the section are definite areas of myxomatous degeneration and others of well-developed fibrous tissue. The diagnosis thus far then is fibromyxoma the typical structure of the majority of innocent false neuromata (Figs 158-159).

Alexis Thomson¹ in his monograph on this subject has collected five cases of solitary

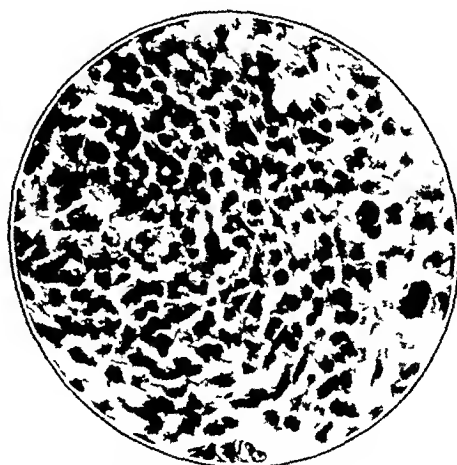


FIG 160—Cyst wall, oil immersion, magnification of highly cellular area showing resemblance to sarcoma.

fibromyxomata from his own clinical experience, and reports five more from the literature

The important practical point about this tumour is as to whether or not it is undergoing sarcomatous degeneration, as these fibromyxomata so frequently do. There are a couple of masses of cells staining deeply with hæmalum and quite definitely of a different



FIG 161—Transverse section of nerve trunk above the cyst. Stain hæmalum and eosin. Shows also fully normal nerve tissue.

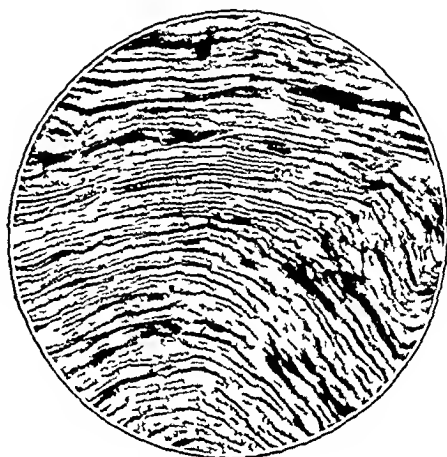


FIG 162—Longitudinal section of nerve trunk below the cyst. Stain hæmalum and eosin. Shows normal nerve tissue.

type from the fibroma cells of the main tumour. Powell White does not consider that they are sarcomatous, but does not explain their presence. Fig 160 shows a view of one of these areas under an oil-immersion lens. A few of the cells are undoubtedly lymphocytes, and perhaps we have here merely a small lymphatic vessel packed with lymphocytes. It would appear, therefore, that though this tumour was essentially innocent in its inception, the presence of possible early sarcomatous change cannot be denied, and that, although the prognosis would appear good from histological evidence, the case still requires very careful observation.

2 Transverse section of nerve above tumour—Fig 161

This, except for a slight increase of interstitial fibrous tissue, is perfectly normal.

3 Transverse section of nerve below the lesion—Fig 162

This is also perfectly normal histologically, except for two or three small masses of polymorphonuclear leucocytes found lying in between the fibres of one nerve bundle. The portion of section shown in the microphotograph is longitudinal to the long axis of the nerve, but shows perfectly normal tissue.

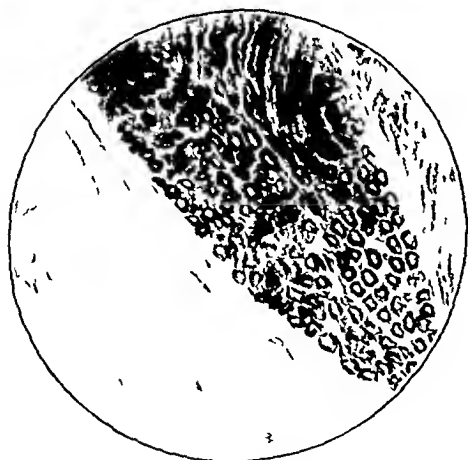


FIG 163—Transverse section of nerve-trunk below the cyst. Stain by Weigert's method. Shows perfect myelination.

The Weigert section shows no sign whatever of any myelin degeneration (Fig 163).

The sections above and below the cyst would, then, tend to confirm the view of the innocence of the tumour, and the perfect Weigert staining confirms the lack of any clinical evidence of loss of conduction.

CONCLUSIONS

1 That a hæmorrhagic cyst of spontaneous origin arising in a peripheral nerve may be innocent

2 That, in the absence of more definite evidence of sarcoma, such as infiltration of the nerve above and below the lesion or adherence to surrounding structures, it would have been advisable in this case merely to puncture the cyst and remove as much as possible of its wall without interfering with the continuity of the nerve bundles, thus avoiding the risk of incomplete regeneration after resection and end-to-end suture

HISTORICAL SURVEY

The occurrence of solitary fibromyxomata of peripheral nerve-trunks, though comparatively rare, is probably not extremely so. The subject does not seem to have been at all completely reviewed since Alexis Thomson¹ published his classical monograph in 1900. This surgeon is, however, able to describe five cases of his own, and has collected five cases from the literature up to that date. One of these latter presents features closely resembling the case described above, and perhaps merits transcription here — 'This case is recorded by Surgeon Lieutenant-Colonel Hatch. A healthy-looking man, age about 40, complained of a tumour at the back of the left thigh of ten years' duration. At first it gave him no inconvenience. About a month before admission to hospital it became slightly painful. A tumour the size of an orange was found at the back of the thigh at the junction of the middle and lower thirds. The skin over it was tense and shining. The swelling was globular, firm, smooth and slightly elastic, very movable from side to side but not up and down. If the leg was flexed the tumour could be moved more readily. The limb was not wasted. Pain was felt at the site of the tumour and down the leg while walking, which made the patient limp a little. There was no tingling sensation. The femoral glands were not enlarged. All the organs were healthy. A longitudinal incision 5 in long was made over the tumour, the flexor muscles were dissected off the surface. The tumour was slightly lobulated and had a bluish appearance, towards the lower end a yellow spot was seen, and here there was slight bulging. Continuing the dissection up and down, the swelling was found to be connected with the sciatic nerve, a few veins and nerve fibrils ramified over the surface. A small puncture accidentally made on the surface allowed pure blood to escape with considerable force, the opening was closed with forceps, and the nerve fibrils which were in contact with the surface were very carefully separated on both sides. The sac was then completely evacuated and the trunk of the nerve which was spread out on its deep and anterior surface was defined and the sac removed. A skein of fibres which had been cut near the upper end of the cyst was sutured. The sac had a thin wall, apparently continuous with the sheath. It contained pure blood, a little fibrin, and hæmatoidin crystals. Recovery was quite satisfactory.'

Unfortunately no histological examination is reported of this case. The decision to perform a conservative operation must also have been easier here as the cyst was not so intimately surrounded by nerve fibres as in the present case.

Thomson only reports one other cyst, a case of Zum Busch's. This was a lesion of the ulnar nerve in a patient, age 30, who had had two injuries to the elbow, a probable fracture of the lower end of the humerus in childhood and a more indefinite injury five weeks before he came under observation. There were signs of ulnar paralysis, and the cyst was found to contain yellowish, jelly-like material resembling synovial fluid. The cyst was evacuated and its walls were sponged with corrosive sublimate. The paralysis disappeared and the patient resumed his occupation of furniture polishing in two months after operation.

Here again apparently no histological diagnosis was attempted, and it is quite open to doubt as to whether the lesion was neoplastic or traumatic in its origin.

In the course of a search through the literature since 1900 I have not found any

report of a solitary cystic fibromyoma of a peripheral nerve. The characteristic tumour of this type, as one finds it in the generalized form described by Von Recklinghausen, is solid in consistence.

Solitary fibromyomata have been described by Foote³ and by Gatch and Ritchey,⁴ two specimens being reported by each. Three of these arose in relation to the brachial plexus, and are of interest in that they were large masses of new growth spreading irregularly between muscle planes, but showing their innocence by absence of invasion of muscle or other tissues. Kerr² in 1914 described what he believed to be the largest fibroma on record. It was a pure fibroma arising from the sciatic nerve, weighed 1 lb 3 oz, and measured 6 in by 3 in.

The histological diagnosis of such tumours—with reference to innocence or malignancy—seems to have presented difficulties before now. Foote reports that his first case was returned by the histologist as sarcoma, but the course of this case and the histological examination of his second case led to further sections being taken of the first, with the result that both his tumours were eventually considered to be innocent in type.

As regards the etiology of fibromyomata, some observers consider that trauma plays an important part in their inception. They base this opinion on the fact that it is in the nerves which are most exposed to slight repeated traumata that these growths arise. Common opinion seems to be that the median is the nerve most frequently attacked. The sciatic also seems a favourite site.

Recurrence after operation is rare, and seems thoroughly to justify a conservative line of treatment in the absence of any definite macroscopic signs of malignancy. Foote mentions an interesting case reported by Bruns in which the surgeon, after the successful removal of an innocent tumour of a nerve in the foot, was required to operate on a similar growth in the sciatic of the same limb.

The absence of any loss of motor or sensory power of the nerve involved in innocent fibromyoma seems to be universally agreed upon, the symptoms in the most severe cases being a variable, but generally slight amount of tingling and 'pins and needles' in the sensory distribution of the nerve.

My thanks are due to the following for facilities and assistance in the report of this case: Mr E. E. Hughes, Professor J. S. B. Stopford, Dr Charles Powell-White, and Mr Harry Platt.

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- ³ FOOTE, EDWARD W., 'Two Cases of Solitary False Neuroma—probably Nonmalignant', *Amer. Jour. Med. Sci.*, 1910.
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CONGENITAL DIAPHRAGMATIC HERNIA.

By J B HUME, London

A REVIEW of the literature on this subject would show a wide divergence of opinion on the etiology of the various types of diaphragmatic hernia, and on the treatment that should be adopted. The scope of this paper is to attempt to define, first, the various types from the standpoint of the pathological anatomist, and to offer an explanation of their mode of origin, and secondly, to indicate the lines on which surgical treatment may be attempted. A complete report of one case is included and reports on the examination of two other specimens of the condition.

DEVELOPMENT OF THE DIAPHRAGM

The diaphragm arises by modification of the septum transversum of the early embryo. This septum is mainly a mesoblastic vehicle for the ducts of Cuvier from the body wall to the heart. It occupies an oblique plane, sloping downwards and forwards from the cervical region, immediately dorsal to the heart. From this position it makes a gradual descent, reaching its final level about the third week of intra-uterine life.

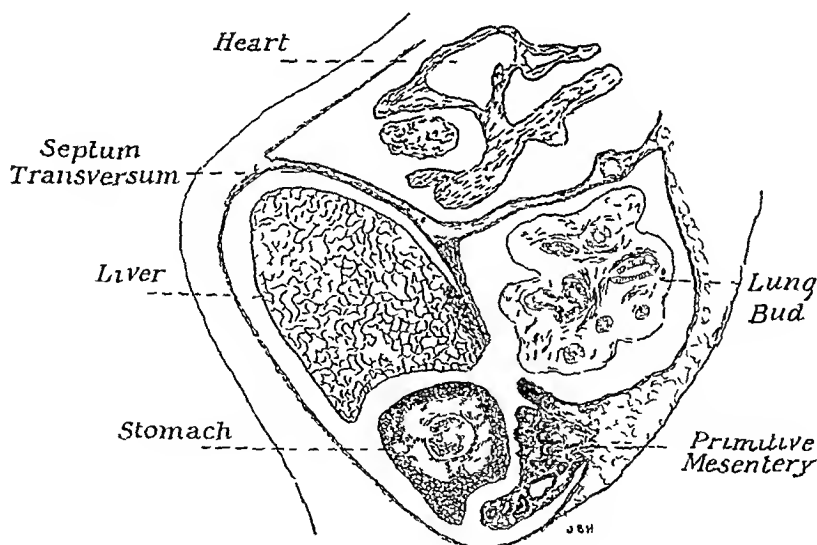


FIG 164—Sagittal section of 11 mm embryo through the hiatus pleuroperitonealis (½ obj)

During the third week it has a posterolateral opening on either side, the hiatus pleuroperitonealis through which the lung buds pass upwards, as the septum transversum descends, the pleural cavity thus being an extruded portion of the coelom (Keith). As the septum transversum descends, mesoblastic cells in the lower dorsal region, representing the cephalic end of the primitive mesentery, proliferate and, bridging across the opening, establish a connection with the septum transversum (Fig 164).

The diaphragm is thus constituted of a ventral portion derived from the septum transversum, and a dorsal portion derived from the primitive mesentery. In the primitive mesentery, near its free border, is placed the developing oesophagus and stomach.

In the fourth week the hiatus pleuropertonealis is closed by a double fold of pleura and peritoneum. Muscle fibres are now present in the septum, but not in the membrane closing the hiatus. This can be seen macroscopically from the fifth to the twelfth week as a transparent triangular area situated between the costal and spinal muscle origins (Fig 165)

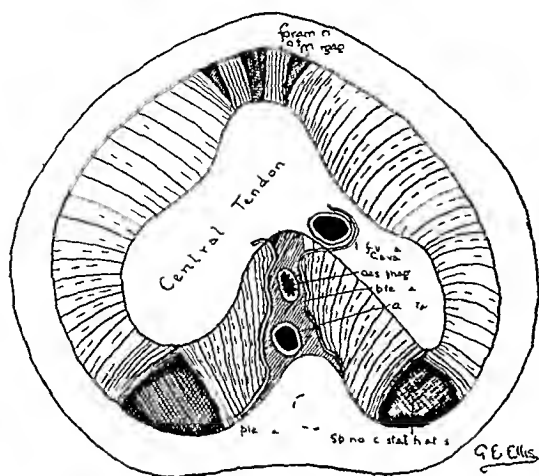


FIG 165—The thoracic aspect of the diaphragm, showing the position of the hiatus pleuropertonealis (spinocostal hiatus)

The thinnest portion of the rest of the diaphragmatic sheet is the central area of each dome. The right dome is completely filled by the liver, while the left contains a small portion of liver, the stomach, and coils of intestine of the proximal and distal loops of the mid gut, which are undergoing rotation about the axis of the vitelline artery. The grouping of intestine in this region is due to the fact that this is the most roomy part of the abdominal cavity, the anteroposterior diameter of the lower abdomen being much less, and the pelvis merely a potential space.

Mode of Origin of Congenital Diaphragmatic Hernia—It is probable that this arises at the period of development just described.

TYPES OF CONGENITAL DIAPHRAGMATIC HERNIA

Four types may be described (I) *Hernia through the hiatus pleuropertonealis* (II) *Hernia through the dome* (III) *Hernia through the oesophageal orifice* (IV) *Absence of the left half of the diaphragm*.

Type I Hernia through the Hiatus Pleuropertonealis—This is, if foetal cases are included, the commonest type. It is, however, rarely met with in adult life. Keith collected 21 cases from the medical museums of London, and of these only 2 survived more than a few weeks after birth. It is suggested that the intestine follows the lung bud through the hiatus at the time when the former leaves the coelomic cavity, or that it passes through from lack of intra-abdominal space, before the pleuropertoneal membrane closes the hiatus. That it is a fact that the hernia occurs at an early stage is shown by the opening retaining the shape of the hiatus pleuropertonealis, and by the absence of any sac.

The admitted greater frequency on the left side is due to the mass of liver protecting the right hiatus. The lung, on the affected side, is partially under developed in all cases. The intestine is invariably in a condition of incomplete rotation, and it is common for the greater part of both large and small intestine to be intrathoracic.

Type II Hernia through the Dome—This may occur as a true hernia (in other words, a hernia with a sac), or as a false hernia.

Pressure of abdominal organs on some weak point of the diaphragm may produce a hernia, either before or after birth. Such cases, in which sacs of thinned-out peritoneum and diaphragm were present, have been described by Lawrence and Petit. A generalized bulging of one dome, with a consequent abnormally high situation, is described by radiologists, and named 'eventration' (Sailer and Rhem, Bayne Jones). It is debatable whether this should be classed as a hernia at all.

The above explanation may be true of some cases, but certain facts suggest an alternative explanation now brought forward. The herniated structures are usually the stomach and omentum, or, in addition, a portion of the transverse colon, with perhaps coils of small and large intestine as far as the splenic flexure.

The arrangement of the large intestine is important, it is found to the left and partly in front of the small intestine, in the position occupied before the normal axial rotation of the gut is completed. The inference is that the hernia occurred at the time when the rotation was taking place (that is between the fifth and eighth weeks), either by the gut becoming included in the junction between the dorsal and ventral portions of the diaphragm, or by pressure and movement causing the primitive diaphragm to give way. Incomplete rotation of the gut thus favours the formation of hernia, and the herniation of the gut obviously brings rotation to an end. In this sense incomplete rotation is at the same time the cause and effect of a diaphragmatic hernia.

Case 1 illustrates this, while cases in which this condition is described are reported by Beekman and Duval. The latter records repeated attacks of appendicitis occurring in a boy, age 12, with a diaphragmatic hernia. The symptoms were referred to the chest, and the appendix was found below the left clavicle.

A partially detached lobe of the liver is a frequent content of hernias through the dome. Keith records it in several foetal cases, and Monks, at an autopsy on a man of 43, who died of pneumonia, found a portion of the left lobe, together with the stomach and transverse colon. Oui and Devulder found two openings, one in each dome, in a full-term male foetus, abnormal lobes of the liver being responsible for both. The liver was in three portions, one in the right side of the thorax, a central abdominal portion, and one in the left side of the thorax. The liver in its development expands into the substance of the septum transversum and these cases are explained by a complete penetration of the septum having taken place, leading to a corresponding deficiency of the diaphragm. The stomach developing immediately posterior to the septum, probably becomes included in the attachment of the primitive mesentery to the septum transversum.

Type III. Hernia through the Œsophageal Orifice.—As in the dog fish, in the early human embryo the stomach occupies a retro-pericardial position. In the later stages of development in the mammalia, a migration of the stomach towards the tail occurs, accompanied by a corresponding elongation of the œsophagus. In the human embryo this migration is almost complete before the final

constitution of the diaphragm. When the tailward migration fails, the stomach instead of the œsophagus is found in the position of the normal œsophageal opening in the diaphragm. This opening is in consequence enlarged to approximate to the calibre of the stomach, though some degree of constriction is usually present. A portion of the ectom becomes separated off, and encloses the stomach in a peritoneal sac. As the stomach completes its descent it carries this portion of peritoneum down with it and a remnant of it may be seen in the foetus as a small finger-like process, to one or both sides of the œsophagus, and named the para-œsophageal recess. A small portion of stomach or intestine may pass up into one of these peritoneal diverticula. Case 2 (Fig 166) illustrates this failure of migration, the stomach lying in a sac in

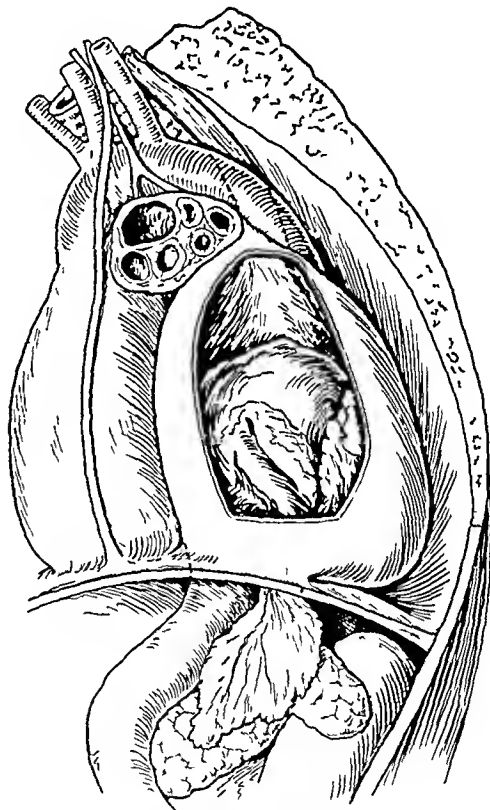


FIG 166.—Hernia through the œsophageal orifice. Lateral view semi-diagrammatic. The dotted lines show the termination of the œsophagus.

the posterior mediastinum, and the œsophagus ending just below the bifurcation of the trachea

One other extreme case has been recorded by P Bailey, while Huffman described a true hernia in the same position, the œsophagus running along in the posterior wall of the sac for a short distance, before entering the stomach 3 cm above the diaphragm. A case operated on by Matthews and Imboden is probably similar.

The œsophageal opening is normally oblique, and placed in the thick muscular portion just above the crura. Hernia through this orifice is more likely to be due to these developmental causes than to a gradual stretching from increased intra-abdominal tension, whilst trauma is more likely to affect the costal region.

The present unsatisfactory subdivision between congenital and acquired (or traumatic) diaphragmatic hernia is largely due to the fact that authors assume a hernia occurring through the œsophageal orifice to be an acquired one. Bevan states that "acquired hernias of the diaphragm always occur at the œsophageal orifice, just as inguinal hernia occurs at the external abdominal ring, the œsophageal orifice being the normal weak point in the diaphragmatic wall". Lawford Knaggs reports 8 such cases, 6 of them having a definite sac, which he adds, "is conclusive evidence that the rupture has been acquired", and in the other two cases says, "but since the hernia took place through a dilated œsophageal orifice in each case, there is a strong presumption that both were acquired". Such a view is scarcely tenable if the embryology above described is accepted. *Case 2* is strongly illustrative of this point.

Type IV—Absence of the Left Half of the Diaphragm—Complete absence of the left half of the diaphragm is not infrequently found in fetal cases. The whole diaphragm to the left of the œsophageal opening and the left crus is absent. Parietal pleura and peritoneum form a continuous sheet. Abnormal lobes or semi-detached portions of the liver are frequently found, and the greater part of the intestine, in a partially rotated condition, lies in the chest. (See *Case 3* and *Figs 168* and *169*.) Such a condition is almost always incompatible with life, though it is recorded that one boy lived to the age of 17 (Beckman).

Unusual Forms of Diaphragmatic Hernia—Hernias have occurred through other openings in the diaphragm, but are of extreme rarity. The whole literature contains but 8 recorded cases of hernia through the foramen of Morgagni—the small interval for the passage of the superior epigastric artery between the sternal and costal slips. A hernia accompanying the sympathetic trunk under the internal arcuate ligament is twice recorded. Hernia into the pericardium has also been met with (Stoeber).

Occurrence—The writer has collected from the English, French, and American publications 35 cases of undoubted congenital diaphragmatic hernia during the years 1910 to 1921, many of which are quoted in the text. Previously, Balfour in 1869, Lawford Knaggs in 1904, and Keith in 1910, have published collected cases of diaphragmatic hernia. The 35 cases fall into the types above described as under—

Type I—HERNIA THROUGH THE HIATUS PLEUROPERITONEALIS 1 case

A full-term fetus

Type II—HERNIA THROUGH THE DOME 18 cases

True hernia (i.e., with sac) 6 cases, False, 9, Not stated, 3

Right dome, 2 cases, Left, 16

Age incidence—20 hours to 20 years, 6 cases, 20 to 40 years, 1 case, 40 to 55 years, 11 cases

Incomplete rotation of gut 3 cases

Type III—HERNIA THROUGH THE ŒSOPHAGEAL ORIFICE 12 cases

True hernia, 6 cases, False 2, Not stated, 4

Age incidence—7 to 20 years, 4 cases, 20 to 40 years, 1 case, 40 to 60 years, 5 cases, 60 to 77 years, 2 cases

Type IV—ABSENCE OF LEFT SIDE OF DIAPHRAGM 4 cases

Fœtal cases, 3, 1 boy, age 17

Clinical Course and Complications—In the great majority of these cases, those discovered accidentally after death from some other cause being excepted, there were symptoms referable to some degree of obstruction. This was less marked in *Type III* than in *Type II*, and in some cases only appeared from time to time.

An unusual number of complications were met with in the latter type. Mention has already been made of a case of chronic inflammation of an intrathoracic appendix. Meyer reports a case of volvulus of the stomach (Willett reported one in 1897), Stewart, of strangulation of the intestine, Lennox Gordon, of perforation of a gastric ulcer into the right thoracic cavity, and Mercade, of perforation of a herniated stomach by a gunshot wound.

The only complication amongst the œsophageal type was one case of tetany (Greig).

Notes on Surgical Treatment—Surgical treatment is unlikely to be required for hernia through the hiatus pleuropentonealis (*Type I*). In those cases that survive more than a few days, the opening is large enough to prevent obstruction. Under certain circumstances such a condition might demand a herniotomy, but no attempt would be made to transfer the intestine to the abdominal cavity.

All cases in which the condition is suspected should have a radiographic examination made during the passage of an opaque meal. Reference to the relative positions of the œsophagus and the stomach shadow will show the situation of the opening in the diaphragm. If some hours later, the meal is seen in an intrathoracic large intestine, it is improbable that any surgical procedure, beyond enlargement of the orifice as a measure in cases of obstruction, would be of any avail.

In cases in which some operative procedure is decided on, an extensive thoracotomy, by removal of a large portion of the 7th rib and wide retraction of the parts, would give an excellent exposure of the upper surface of the dome and a view of the thoracic contents.

Dilatation of the intrathoracic portion of the stomach sometimes calls for operation particularly this has occurred in cases of *Type III*. The stomach should, if possible be emptied by means of an œsophageal tube, reduced as far as possible from the thoracic aspect, and its margins sutured to the opening in the diaphragm (Beckman, Downes). This is at best a palliative measure, another that has been tried is suturing the viscus to the anterior abdominal wall, through another incision (Mathews and Imboden).

In cases of *Type II* when only a small portion of the stomach is herniated and can be readily reduced, an attempt may be made to close the orifice by suture. Some authors report a measure of success with this procedure (Frank).

NOTES OF CASES

Case 1—Diaphragmatic hernia illustrative of *Type II* with incomplete rotation of the intestine (Published by kind permission of Dr H. H. Tooth)

CLINICAL HISTORY—H. T., a boy, age 6, was admitted to hospital on account of an attack of acute abdominal pain. Vomiting followed, and continued for 48 hours. The temperature was 97°, the pulse 104, and respirations 40. There was no history of any comparable attack. Six months previously he had been knocked down by a vehicle and sustained concussion, being unconscious for fourteen days.

The physical signs were those of a left-sided pneumothorax: succussion splash, metallic sound and bell sound all being present. The heart was displaced to the right, its left border being behind the sternum. The percussion note was impaired below the angle of the left scapula. The metallic and bell sounds varied from time to time, sometimes being present and sometimes absent.

The report of a *ri* examination was "complete pneumothorax, left side, the left diaphragm considerably higher than the right." Paracentesis thoracis was performed in the 5th left intercostal space, air bubbled out, and some brownish material was withdrawn. Four hours later the patient suddenly died.

POST MORTEM—Fig. 167 illustrates the arrangement of parts in the specimen obtained. A circular opening 2½ inches in diameter was present in the left dome of the diaphragm, to the left and slightly in front of the œsophageal orifice. Its edges were thick, rounded, and muscular and perfectly smooth. The opening was subdivided into two compartments by a horizontal band



FIG 167—Hernia through the left dome The liver has been removed

of adhesions stretching across its left margin to the 8th left costal cartilage (This band forms the anterior boundary of the opening in Fig 167, the anterior compartment having been destroyed)

The stomach passed into the thorax through the posterior compartment, being looped up as in the fatal specimen represented in Fig 168. With it was the ascending colon, the great omentum and the gastrohepatic omentum. The body of the stomach filled the greater part of the left pleural cavity in addition to the marked dilatation, the muscular coats were greatly thickened. It contained over a pint of partially digested food. A constriction was present at the junction of the body and the pyloric portion where it re-passed under the band of adhesions.

A condition of incomplete rotation of the gut was present. The large intestine from the cecum to the splenic flexure lay in a U shaped loop in the thorax, the commencement of the ascending colon and the end of the transverse colon being in the posterior part of the omphale. The apex of the cecum was directed upwards, passing through the anterior compartment (in the figure it is shown drawn over to the left). The appendix lay on the right side of the mesentery of the small intestine, and below the ileocecal junction. The root of the mesentery of the small intestine, spread out fanwise, ended over the lower pole of the right kidney. The head of the pancreas projected above the duodenum, and under the Spiegelman lobe of the liver, from which it was separated by a pocket of the lesser sac of the peritoneum. With this exception the lesser sac was completely intrathoracic.

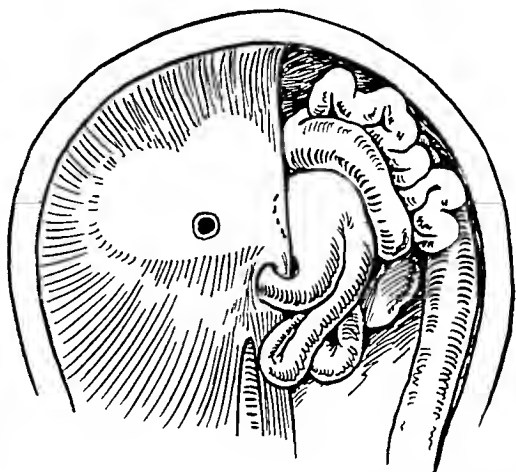


FIG 168—Absence of left half of the diaphragm, abdominal aspect, diagrammatic

The left lung was collapsed and lay with the root in the normal position. Sections showed it to be atelectatic. The pleura and peritoneum were continuous, there were no signs of old or recent pleurisy.

(This and the two following specimens are now preserved in the Museum of St Bartholomew's Hospital.)

Note on Case I—Objection might be raised to this case being classed as a congenital one, in view of the history of trauma. The arrangement of the intestine, however, indicates that it had always occupied this position, the process of axial rotation being arrested in the position seen in the specimen. The fan-like termination of the root of the mesentery, and the reversal of the course of the small intestine present in this case, are characteristic of cases of incomplete rotation, and are strong evidence against the cecum having been drawn up into the thorax. The margins of the opening in acquired cases usually show either scar-tissue or adherent omentum, are frequently irregular in shape, and are more common in the costal zone. In these cases the lung is compressed, and situated in the upper part of the pleural cavity, not as in Case 1, atelectatic and at the lung root.

The second case is that of a man, age 56, brought as a subject for dissection to the anatomical department of St Bartholomew's Hospital. Death was certified as due to bronchitis and heart failure.

Case 2—A hernia occurring through the oesophageal orifice (Fig 166, Type III)

A large opening 2½ inches in diameter, surrounded by thick, rounded muscle, is present in the position of the oesophageal opening in the diaphragm and opposite the 10th thoracic vertebra. Through this opening the peritoneal cavity communicates with a large sac, situated in the posterior mediastinum and containing the body of the stomach and the greater part of the gastrohepatic and gastrocolic omentum.

The sac consisted of thickened peritoneum with external coverings of two layers of mediastinal fascia. Anterior to it was the pericardium, posteriorly the aorta, and to left and right the mediastinal layers of the pleura. The great vessels were not altered in position. The left lung was rather smaller than normal, its lower lobe was compressed. The oesophagus passing down behind the root of the lung ran for a short distance in the posterior wall of the sac before entering the stomach. The celiac axis artery was given off at its normal level, its coronary branch and the two epiploic arteries passed through the opening in the diaphragm in their corresponding

omenta to reach the stomach. No obstruction was present at the junction of the body and pyloric portions of the stomach. The body of the pancreas was bent at an angle opposite the diaphragmatic opening, but did not pass into the thorax.

No other abnormalities were discovered.

Case 3—A foetal specimen illustrating *Type IV*, that of the absence of the left half of the diaphragm.

Fig 168 indicates the extent of the diaphragmatic loss, and shows the stomach passing into the thorax immediately to the left of the oesophageal opening. The fundus is directed upwards and to the right. The opening was partially blocked by an almost detached portion of the left lobe of the liver.

The only portions of the alimentary canal remaining in the abdomen were the duodenum, and the colon below the position of the splenic flexure.

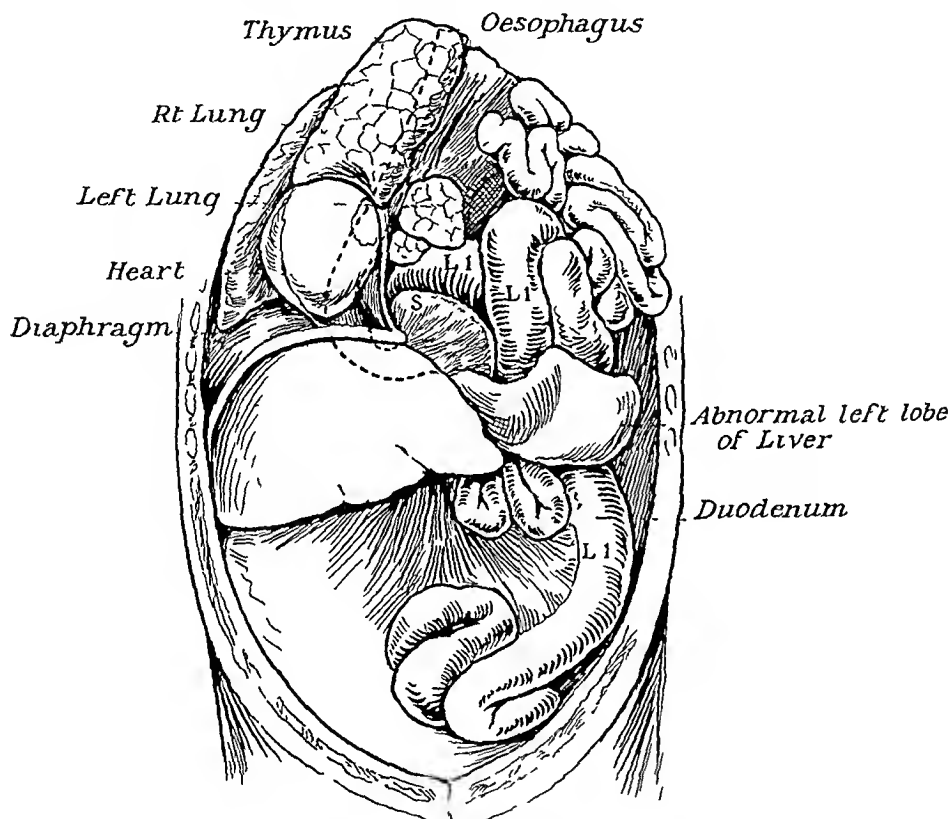


Fig 169—Absence of left half of the diaphragm. The course of the oesophagus is shown by dotted lines. (S) Stomach (L) Large intestine.

Fig 169 shows the colon arching over the small intestine, the cecum being below and to the right. The spleen lay in a sac formed by its passage through the posterior mediastinum into the right pleura.

There was no trace of any diaphragmatic remnant on the left side, the parietal pleura and the peritoneum forming one continuous layer.

SUMMARY AND CONCLUSIONS

1. Diaphragmatic hernia may be classified as—

a. Traumatic (i) true, (ii) false

b. Congenital (i) true, (ii) false

2. Congenital hernia occurs through the left dome most commonly, to a less extent through the oesophageal orifice. In other situations it is but rarely met with in adult life.

3 Hernia through the hiatus pleuroperitonealis takes place before the closure of that opening in the embryo

Hernia through the dome of the diaphragm is due to some primary congenital ectopia of viscera, occurring at the time of formation of the diaphragm, and sometimes associated with incomplete rotation of the intestine

Hernia through the oesophageal orifice is due to failure of the tailward migration of the stomach, or in some cases to the persistence of a para-oesophageal recess

4 Surgical treatment is palliative only Thoracotomy is the route of election

My thanks are due to Sir Arthur Keith, Professor G E Gask, and to my colleagues Drs T H G and L R Shore, for valuable assistance and advice in the preparation of this paper

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RE-SUTURE OF PERIPHERAL NERVES

By JOHN S B STOPFORD, MANCHESTER

We are confronted from time to time with patients who have received no benefit from the suture of a peripheral nerve, and it is then necessary to decide whether, in the case of certain nerves, to advise the patient to submit to re-suture, or to consider the condition as irreparable. This subject is of greatest importance in connection with the ulnar or median nerves since in the case of the musculospiral or even the sciatic nerve alternative orthopedic measures offer such good functional results that a re-suture rarely needs to be entertained. Information about the prognosis after re-suture of a nerve is so scanty that at present it is not easy to decide when such a procedure is justified, and therefore it may prove helpful to record a series of cases in which this operation has been performed, to see if any serviceable deductions may be drawn from an analysis of them.

It is not intended in this paper to discuss the obvious contra indications for re suture, as irreparable arthritic changes or contractures, nor to mention technical points in the operation of suture of a nerve, but rather to discover the principal factors which influence regeneration and the subsequent occurrence of a good functional result.

During the last five years I have had the opportunity of observing the results of re-suture in 14 patients, the nerves affected being median 5, ulnar 7, musculospiral 1, external popliteal 1. In all except one of these (No 2 in the following table) it has been possible to keep the patients under observation for a sufficiently long period to be able to see the end-result. The principal features to be noticed in the fourteen patients are recorded briefly in the table on next page.

From an analysis of this series it is clear that several important factors bearing upon the prognosis have to be considered, and the more significant of these will be discussed in rotation.

1 *The interval which has elapsed between the reception of the injury and the date of the re-suture*

From the present series it would appear that this is an important factor, since out of 7 patients in which the interval exceeded two years there were 6 complete failures. In all six failures the interval was approximately three years or longer. In a large series of 271 cases of secondary suture¹ the opinion was formed that a delay *per se* of twelve to eighteen months had no appreciable effect upon the extent of recovery. If the interval exceeded that time the prognosis was not so good when the suture had been performed in the distal part of the limb, whereas, in the proximal part, a delay of two or three years did not prejudice the chances of success. The number of re-sutures is too small to judge the influence of level, but in other respects the results seem to support the conclusions arrived at from the investigation of a large number of cases of secondary suture.

It is of interest to notice that the four best results were obtained in patients where the delay was between twelve and eighteen months.

Re-suture does not appear to be a hopeful procedure if three years have elapsed since the time of the injury, a suggestion as to the cause for a graver prognosis after the longer delay will be made at a later stage.

2 *The cause of failure after the original suture*

It is not always possible to determine this, but it is obvious that the original cause must frequently have an important influence upon the prognosis after re-suture. In two patients the two extremities of the nerve had broken apart after the first operation, and in another the failure was probably due to faulty technique, conditions which cannot in

themselves prejudice the success of a re-suture. In one case the original operation consisted of the insertion of a nerve-graft, this case has been included, although strictly speaking the second operation cannot be called a re-suture, since the general conditions are so similar to those prevailing in the rest of the series. In 6 others the failure was due apparently to intraneural fibrosis, which is usually most severe and extensive after sepsis, but may occur irrespective of this. In five of the six patients in which a failure is recorded after re-suture, intraneural fibrosis seemed to be responsible for the failure after the original suture.

TABLE GIVING RESULTS OF RE-SUTURE IN 14 PATIENTS

Not — PRT = Pronator radii teres FSD = Flexor sublimis digitorum FPD = Flexor profundus digitorum
FLP = Flexor longus pollicis, FCU = Flexor carpi ulnaris, ADM = Abductor minimi digiti

CASE NO.	NERVE INJURED	SITE OF INJURY	INTERVAL BETWEEN DATE OF INJURY AND RE-SUTURE	PROBABLE CAUSE OF FAILURE AFTER FIRST OPERATION	RESULT OF RE-SUTURE
1	Musculospiral	Arm	40 months	Sepsis and intraneural fibrosis	Failure
2	Median	Elbow	13 months	Separation of ends	PRT, FSD, FPD, FLP show voluntary power. Slight recovery of analgesia
3	Median	Forearm	33 months	Intraneural fibrosis	Failure. Amputation of hand subsequently
4	Median	Wrist	26 months	?	Recovery of analgesia and some recovery of anesthesia
5	Median	Arm	14 months	?	All muscles show voluntary power. Recovery of analgesia
6	Ulnar	Arm	14 months	?	FCU, ADM, interossei show voluntary power. Recovery of analgesia
7	Ulnar	Arm	14 months	?	FCU, FPD, ADM show voluntary power. Some recovery of analgesia
8	Ulnar	Forearm	34 months	Bad technique	Failure
9	Ulnar	Elbow	35 months	Intraneural fibrosis	Failure
10	Ulnar	Wrist	21 months	Intraneural fibrosis	FCU, FPD show voluntary power
11	Ulnar	Arm	17 months	Sutures broke away	FCU, FPD show voluntary power
12	Median	Forearm	72 months	Intraneural fibrosis	Failure
13	Ulnar	Forearm	23 months	Nerve graft	FCU, FPD show voluntary power. Recovery of analgesia
14	External popliteal	Thigh	15 months	Intraneural fibrosis (6 inches resected at re-suture)	Failure

In all at the operation of re-suture, an extensive length, averaging two to two and a half inches, was excised in order to attempt to get above the most severe fibrosis, and in one (No. 14) where very marked intraneural changes were encountered, Mr. Platt excised six inches.

From a histological study of excised pieces of nerve and more extensive examinations of the proximal part of the nerve in irreparable cases where amputation has been found

necessary, quite severe intraneural changes have been traced as far as eight inches above the level of the lesion, and it seems probable that after sepsis they may extend to the spinal cord. From my own observations, I am of the opinion that one of the most frequent causes of failure after suture of peripheral nerves, following gunshot wounds, is the fibrosis in the nerve-trunk, around bundles and even within bundles surrounding the individual nerve-fibres. In an appreciable proportion these intraneural changes are so extensive that the most liberal resection practicable fails to get above them.

3 'Bad shunting'

The risk of efferent fibres growing down to afferent terminals or vice versa, which with the most perfect surgical technique must occur to some extent in almost all secondary sutures, is still greater after re-suture, since a greater length is excised under the latter conditions, and the intraneural anatomy is still more disturbed. This factor is of less importance in the case of the musculospiral nerve, which is so largely composed of efferent



FIG. 170.—Section of upper sacral region of spinal cord of a rabbit 29 days after division of crural nerve in the gluteal region. Note chromatolysis and feeble staining of most of the cells in interior cornu at (A).

(The section is from an experimental investigation at present in progress which has been aided by a grant from the Royal Society.)

fibres, but is of great significance in the median or ulnar, in which nerves afferent, efferent, and sympathetic fibres are in more equal proportion. We are liable to imagine that there is slight change in the intraneural pattern as a nerve extends to its distribution, and that the bundles formed in the proximal part of the nerve persist more or less distinct with only slight exchange of fibres as they pass to their destination, yet Langley and Hashimoto² and Compton³ have shown conclusively that all nerve-trunks in the limbs have an internal nerve plexus before they give off branches. In the large nerves which are considered in this paper the internal plexus involves the bundles and not merely a few nerve fibres, but from the observations of Compton it appears that these plexuses are composed largely, if not entirely, of motor fibres.

These anatomical studies do not minimize the importance of the adoption of every possible surgical technique to avoid distortion during the performance of a nerve suture, but they do show that, except in a few circumstances, the best technique cannot prevent some bad shunting, and that this factor becomes of greater significance when the resection is extensive.

4 *The effect of a third section of the nerve-trunk upon the cells of the anterior cornu and posterior root ganglion*

This factor has a more theoretical interest than the former three, but nevertheless it must have some practical bearing. It has long been known that injury to an axone induces a reaction in the cell from which it arises, whether the cell of origin be in the anterior cornu or posterior root ganglion. These changes, which consist chiefly of central chromatolysis (Fig 170), swelling of the cell, and excentrication of the nucleus, are more profound if the nerve is lacerated or torn than if it is divided cleanly, and are more pronounced when the injury occurs in the proximal part of the limb than in the distal. Experimental work upon animals has shown that in the former position the changes may be sufficiently severe to cause complete destruction of some cells, whilst in the latter position no very definite reaction in the nerve-cells may be manifest. The stage of solution is apparent within two days, and persists for about three weeks, at the end of which time most of the cells commence to recover slowly. The period of recovery extends over a considerable time, Buzzard and Greenfield¹ state twenty to eighty days, but after amputation of part or the whole of a limb, changes have been found in the cells of the anterior cornu from three to seven months after the operation. In most cases of re-suture these reactions must occur three times, since injury to axones occurs at the outset and with resection of the nerve-trunk at each operation. The initial trauma—since in gunshot injuries the nerve is usually torn and lacerated—is likely to produce the most severe reaction, and if the injury is in the proximal part of the limb the results of the two resections cannot be disregarded entirely.

In the series published in this paper the reaction due to the additional resection does not appear to be of serious practical consequence, since in the eight cases in which regeneration has occurred after re-suture the extent of both motor and sensory recovery compares favourably with the end-results I have been able to observe in a large number of patients after the performance of secondary suture. In the case of the complete failures it is impossible, with any degree of accuracy, to decide whether this factor is partly responsible for the absence of regeneration, but I am inclined to regard the time element and the intraneural condition of the proximal segment as the most important causes of failure. It is possible that the more serious prognosis after a long delay may be due to the factor at present under consideration, since it has been shown experimentally that a greater number of nerve-cells degenerate and disappear after section of a peripheral nerve if union of the two ends does not take place, and that the recovery of cells in the anterior cornu and posterior root ganglia depends upon restoration of the continuity of the nerve-trunk.

I desire to express my gratitude to Mr H. Platt for his co-operation in the work, and the opportunity to study his patients.

CONCLUSIONS

1 Regeneration may occur, under favourable conditions, after the re-suture of a peripheral nerve.

2 The end results after successful re-suture are similar to those observed after a successful secondary suture.

3 The causes of failure seem to be the same as in secondary suture with the addition of (a) Greater disturbance of the intraneural anatomy by the further resection, (b) The

effect of a third injury to the nerve-fibres upon the cells in the anterior cornu and posterior ganglia

4 Excluding complications, re-suture is contra-indicated, (a) When more than three years have elapsed since the time of the reception of the injury to the nerve, (b) When extensive intraneural fibrosis has been encountered at the first operation

5 *The imperfect recovery of function and sensation, which is almost invariably found, even under the most favourable circumstances, after secondary suture or re-suture, is chiefly due to* (a) Disturbance of the internal anatomy of the nerve-trunk, (b) Intraneural fibrosis

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AN INQUIRY INTO THE RESULTS OF THE OPERATIVE TREATMENT OF INTERNAL DERANGEMENT OF THE KNEE-JOINT.*

By PHILIP H MITCHINER, LONDON

For the purposes of this inquiry, all cases of internal derangement of the knee-joint operated on in St Thomas's Hospital during the preceding ten years, i.e. 1912-21, have been circularized and requested to attend hospital for inspection or, if unable to attend personally, to write a truthful account of the present condition of the knee, and a brief history of its progress since operation.

Statistical Notes—The total number of cases of internal derangement operated on in the period covered by the inquiry is 225, to all these cases inquiries were addressed, and replies were received from 134, or approximately 60 per cent. Of this number between 80 and 90 attended for inspection, and were carefully examined as to the physical condition of both joint and limb, subjective and objective symptoms, and in most cases by radiographic examination also. The condition of the other joints, especially with regard to osteoarthritic changes, was noted in every case.

Of the total number of cases, 182 were males and 43 females, or a proportion of four males to one female. It is further of some interest to note that there is no increase of the number of female cases in the last five or six years, which the entry of women into more strenuous occupations might have led one to expect.

Only one fatal case occurred in the series, that of a man from whose joint two loose bodies were extracted, and who three days later developed a suppurative arthritis of the knee, with subsequent pyemia, and death on the tenth day, in spite of the fact that the affected joint was removed almost as soon as suppuration manifested itself.

Classification—In regard to the various types of internal derangement met with, the following classification has been adopted for the purpose of this paper, based on the appearances found in the joint at operation—

Group A—Loose body in the knee-joint

Group B—Torn or displaced semilunar cartilage (only including those cases where actual evidence of lesion was observed at operation)

Group C—Hypertrophied synovial fringes

Group D—No definite intra-articular lesion found

Group E—Torn crucial ligaments

Relative Frequency—The relative frequency of these lesions was found in this series to be as follows—

Total cases 225	{	Group A	39 cases =	17.3 per cent
		„ B	125 „ =	55.5 „ „
		„ C	27 „ =	12.0 „ „
		„ D	30 „ =	13.3 „ „
		„ E	4 „ =	1.8

It will thus be seen that Group B cases were more than the total of all the other groups together and it is to be regretted that a further subdivision of this group into tears of

the semilunar cartilage and of the coronary ligament (with displacement of the cartilage) was not carried out

Operation Incisions—Two operative routes had been used in opening the knee-joint (1) A lateral incision over the affected cartilage, either transverse or vertical in direction (2) A vertical median incision with longitudinal splitting of the patella. Inasmuch as only the former incision was used until 1914 it is perhaps unfair to make a comparison of the results achieved by these two operative routes, the following observations may, nevertheless, prove of interest and value

For the purposes of operation there is no doubt that the transpatellar route allows of a much more efficient view of the joint for exploratory purposes, and furthermore admits of the complete removal of the semilunar cartilages—a point of some importance in view of the fact that in over 5 per cent of cases subsequent trouble resulted from the posterior fragment of the semilunar cartilage left *in situ* after removal of the major part through the lateral incision, trouble which, moreover, was cured by subsequent excision of the remaining portion in many such cases. This remaining portion tended to calcify and act as a source of irritation and frequent effusions into the knee-joint, subsequently forming a starting-point for osteo-arthritic changes, or a source of a loose body.

In regard to the immediate after-results of operation—and this period covers, as is shown later, one of over twelve months' duration—there is no doubt that the knee opened by the transpatellar route is more painful and more liable to effusion, and one might think, therefore, more liable to be the seat of lasting after-complaints, but this is not borne out by our investigation.

The ultimate results are, if anything, more satisfactory after the transpatellar operation, no impairment of movement has been noted, and the risk of subsequent trouble from osteo-arthritic changes is certainly no greater, indeed, in cases where the semilunar cartilage needs removal, it is less—as has been pointed out above—than after operation by the lateral route.

A large number of patients operated on by the lateral route, moreover, complain of complete loss of sensation over the head of the tibia and around the ligamentum patellæ, with, in some cases, tingling and pain on kneeling, a symptom which is still present as long as ten years after operation. Examination shows there is in many cases a definite area of anæsthesia over the inner condyle of the tibia, and a larger area around which only protopathic sensation is present.

These phenomena, although not in any case accompanied by clinical trophic lesions of the skin, are a source of discomfort to the patient, and inasmuch as they are due to section of the internal cutaneous branch of the anterior crural nerve (medial cutaneous branch of femoral nerve) at the site of operation, can easily be avoided by use of the transpatellar route.

Results of the Inquiry—The actual results in the 134 cases from whom replies were received, or who were examined, are appended in the following table, where the results in each group have been shown under the following headings—

1 *Good*—Where the patient not only has no complaint to make of the joint, and is able to obtain full use on all occasions with no untoward results, but also writes, or uses expressions, with regard to the success of the operation, such as, "The operation was an unqualified success", "I have not had any trouble with my knee since operation", "I have never had cause to think of my knee since", etc.

2 *Fair*—Where the joint, although giving no trouble during the patient's ordinary everyday life, yet causes pain or has an effusion as the result of over-exertion.

3 *Unsatisfactory*—Where the joint is in the same condition as before operation is a continual source of trouble from pain and effusion or is even worse than before operation.

It may be urged against this method of classifying results that no account has been taken of the actual results of clinical examination, and in reply to this it must be admitted that several cases in which a certain amount of post-operative osteo-arthritic change was present have been included under the heading 'good'. The defence is that such patients made no complaint of this condition up to the time of, or at, inquiry, and since the

result from the patient's standpoint is most important, it was considered that this was the most satisfactory method of classification. A further point in its favour is that only 87 of the 134 cases included actually presented themselves for examination.

TABLE SHOWING THE RESULTS IN EACH GROUP

GROUP TOTALS	GOOD		FAIR		UNSATISFACTORY	
	Number	Per cent	Number	Per cent	Number	Per cent
<i>Group A</i> , 23	14	60.9	5	21.7	4	17.4
<i>B</i> , 74	57	76.7	14	19.2	3	4.1
<i>C</i> , 16	6	37.5	7	43.8	3	18.7
<i>D</i> , 18	5	27.7	3	16.8	10	55.5
<i>E</i> , 3	2	66.6	1	33.3	—	—
TOTAL 134	84	62.7	30	22.4	20	14.9

It will thus be seen that the percentage of good results in the series is 62.7, fair 22.4, and unsatisfactory 14.9, moreover, the best results (76.7 per cent good in *Group B*) were obtained where definite lesions of the semilunar cartilages were present, and the worst (55.5 per cent unsatisfactory in *Group D*) where no definite lesion was observed, and where—in spite of the joint being apparently normal—the semilunar cartilage was excised at operation (see remarks on *Group D* below).

The joint was definitely worse in two cases only. In one where a loose body had been removed from an osteoarthritic joint in 1912, there was now very marked osteo-arthritis, and seven loose bodies in the joint. It may be remarked in passing that this patient was regularly employed in carrying loads up ladders, and was in the habit of 'putting his knee in again' when it locked, which it did frequently! The second case was one in *Group D* where, in spite of no definite lesion being seen, the internal semilunar cartilage had been removed, there was a complete flail joint, due to laxity or atrophy of the crucial and other ligaments and the joint could be dislocated in all directions, the limb was much wasted, and the man could only get about when using a knee-cage.

Notes on Cases in the Various Groups

Group A—There was nothing particular to note in this group save perhaps the greater tendency of these cases to develop osteoarthritic changes.

Group B—Of these cases, 69 showed lesions of the internal and 4 of the external semilunar cartilage—a proportion of approximately seventeen to one in favour of the internal cartilage in frequency.

Group C—It is interesting to note that, subsequent to operation, 5 cases developed tuberculous disease either in the joint itself or elsewhere in the body, and that in all these cases the joint still gives trouble.

Group D—Three cases in this group developed renal disease, and one diabetes, and it seems probable that in these cases the synovial thickening was of a toxic nature, and comparable to the toxic œdema of shins met with in such cases. One case developed acute nephritis immediately after operation, and in this case the change was assuredly of this nature, this man now reports that at present both his renal and knee conditions are quiescent, but that exacerbations of renal trouble are frequently accompanied by swelling in the knee-joint.

It is, furthermore, a noteworthy fact that although amongst the 18 cases traced in this group in only 4 was the surgeon content to close the joint on nothing abnormal being found, yet these 4 cases are included among the 5 'good' results. The remaining 'good' case was one in which hypertrophied synovial membrane only was removed. The remaining 13 cases in which the internal semilunar cartilage was partly or entirely excised have all given subsequent trouble.

Group E—Only 3 of the 4 cases comprising *Group E* can be traced. The fourth one

was of great interest, in that wire loops were inserted to replace a hopelessly disrupted posterior cruciate ligament, and it is to be regretted that this case cannot be traced.

Among the whole 225 cases only 2 joints suppurated, one being the fatal case quoted elsewhere, and the other following a hæmātoma in the joint. This latter case was treated by gauze packing daily, and now, three years after operation, the patient is able to follow his occupation, and has a useful limb with about 50 per cent movement at the knee joint and only very slight lateral mobility.

Clinical Notes on Cases Examined — Post-operative osteo-arthritis changes, or aggravation of osteo-arthritis changes noted in the joint at operation, as judged by comparison with the opposite knee-joint, were noted in 50 per cent of the cases examined, it is, however, of interest to find that in many cases where marked signs of such changes were present, the patient neither complained of, nor had noted, any disability in the joint, indeed, in only about 12 per cent of these cases was any complaint made by the patient. In no case was the change sufficient to cripple the patient entirely.

In several cases, where no complaint was made, a ray examination showed linear calcification in the remains of the posterior part of the internal semilunar cartilage, which is therefore a presumable source of trouble.

It was not found that the cases treated by the transpatellar route showed any greater percentage of arthritic changes, or any greater severity of changes, than those met with in joints opened by the lateral incision, although several of the latter type of cases dated back to 1912 as against 1914 only for the former.

Pain and effusion in the joint subsequent to operation, occurring in attacks and following unusual or undue exertion on the part of the patient, was noted or complained of in nearly all cases operated on in the last three years, i.e., 1919-21. This led to inquiry being made among all the other patients operated on at earlier dates where no such complaint was now made, and it was elicited that in the majority of these cases, now quite cured, pain and effusion in the knee-joint had occurred on exertion for from two to three years after operation and that in many cases this effusion had taken considerable time to be absorbed. Such unwelcome occurrences became less frequent towards the end of the second year after operation, and then ceased, so that from the third year onward the patient could indulge in all forms of exercise without any untoward results.

In those cases where the joint had been fixed by splinting for any length of time after operation, there was frequently marked and persistent muscular wasting around the joint, and in all such cases the convalescence had been much prolonged, and the period during which attacks of pain and synovial fluid effusions were liable to occur was protracted, or even permanent.

Only in the case where hæmarthrus and subsequent suppuration supervened, was any ill effect noted from non-fixation of the joint after operation, and early movement and light massage or faradic electrical treatment seemed to have benefited the patients in promoting rapid absorption of effusions, amelioration of pain, and normal return of tone to the muscles around the joint.

Lastly, it was found that most of the women, and many of the men (who as policemen had to take part in physical drill), complained of pain when the knee was in extreme flexion, as on kneeling or squatting back on the heels, and inability to start rising from these positions. In all cases where this disability was complained of, there were osteo-arthritis changes in the joint, though in the majority nothing else was complained of except the pain on flexion.

CONCLUSIONS

It would appear that the following conclusions can be drawn from the facts elicited in the examination of the foregoing series of cases:—

1 That as regards the actual route selected at operation, the transpatellar is the most satisfactory in all respects.

2 That no method of splinting is necessary or advisable to secure fixation of the

knee after operation, early movement, combined with electrotherapeutic measures, are beneficial in promoting early absorption of effusions

3 That the full benefit of the operation is not to be expected for from two and a half to three years after its performance

4 That those cases where a definite lesion of the intra-articular structure is present give far more satisfactory results (76.6 per cent in cases of damage to the semilunar cartilage, and 60.9 per cent in those of loose body) after operation than where no definite lesion can be found (37.7 per cent with synovial fringes, and only 27.7 per cent where no lesion is seen at operation)

5 That when nothing abnormal is found on the joint being opened, the best course to adopt is to close the joint without interfering with the intra-articular structures

On reviewing the whole of the facts set forth in the foregoing article, it would seem that operative interference in cases of internal derangement of the knee is justifiable, if not indeed desirable in all cases where a diagnosis of a definite lesion of any of the intra-articular structures can be hazarded with any degree of certainty, and where the consequent disability is sufficient to prevent the patients carrying out efficiently their daily avocation

MULTIPLE POLYPI OF THE STOMACH (GASTRITIS POLYPOSA) WITH THE REPORT OF A CASE

By G. PERCIVAL MILLS, BIRMINGHAM

THERE are three fairly clearly defined types of the so called polyadenoma of the stomach —

1 The single large polypoid tumour, usually growing near the pylorus and frequently becoming malignant. Cases have been reported by Bret,¹ Finnis and Friedenwald,² Gibson and Blake,³ Ledderhose,⁴ Ruggles,²¹ and others.

2 Polyadenoma of the 'Brunnerian' type, the structure of which resembles that of Brunner's glands normally found in the duodenum. This type causes a considerable thickening of the mucous membrane over a limited area, and like the first type, frequently becomes malignant. It was first described by Hryem⁵ and again more fully by Soeet,⁶ who considered it to be due to a congenital malposition of Brunner's glands.

3 A form of polyposis first fully described by Menetrier⁷ in 1888 and called 'polyadenome'. In this type the tumours are multiple, sessile or pedunculated, and usually scattered widely over the surface of the stomach. Their microscopic appearance suggests that they are localized overgrowths of the mucous membrane, and they are seldom associated with carcinoma.

Cases of the first two types are well defined, but the third type has given rise to much controversy, and the true nature of the morbid process is still uncertain. It is frequently referred to as 'gastritis polyposa' or 'diffuse polyposis'. The former term is undesirable, as it suggests an inflammatory origin which is not yet proved, the latter possesses the merit of being non committal.

My attention was directed to the subject by the following case.

Case Report—Mr L, age 60. Engaged in retail trade.

HISTORY—The patient had been in perfect health up to seven months before I saw him. About this period he began to get occasional attacks of diarrhoea, and once or twice he vomited after food. The attacks of diarrhoea alternated with constipation, so that a growth of the colon was suspected. There was no pain. More recently he had suffered from a feeling of nausea after food, and complained "that it did not go down properly". He had completely lost his appetite and had lost considerably in weight. At no time during his illness had there been any pain, and he had only vomited half a dozen times during the seven months.

EXAMINATION—This showed a spare man of the wiry type with obvious signs of recent wasting. He had a regular slow pulse and his arteries were reasonably soft for his age. His tongue was clean. The abdomen showed nothing abnormal, no distention, local swelling or splashing. Per rectum the prostate was a little enlarged.

On screening the patient after a barium meal there was seen the typical picture of pyloric stenosis—a very broad stomach with the pylorus pushed over to the right, and strong deep peristaltic waves producing no elevation whatever. Four hours later three fourths of the barium was still in the stomach.

DIAGNOSIS—Carcinoma of the pylorus.

OPERATION—A paramedian incision was made above the umbilicus. A small, hard, nodular growth was found at the pylorus. The pyloric glands were enlarged but soft, and there were a few soft enlarged glands in the gastrophrenic omentum. In handling the stomach a soft slippery substance was felt inside, about in the middle of the body of the viscus. It was obviously attached to the stomach wall, but only loosely, so that it easily slipped away under the fingers. A polypoid growth, possibly secondarily, was suspected, and the resection was planned to include its removal. The usual partial gastrectomy for carcinoma was done. On dividing the body of the stomach to the left of the palpable polypus it was found that the whole body and fundus of the viscus was studded with small polypoid tumours varying in size from that of a split pea to a cherry. They were soft and velvety to the touch, and, while attached to the mucous membrane, were freely movable on the muscular wall. The smaller ones were sessile and appeared like a localized thickening of the mucous membrane, while the larger ones were pedunculated. They were so numerous that it was extremely difficult to avoid them in dividing the stomach. It was obvious that nothing

short of a subtotal gastrectomy would secure their removal, and this was not attempted. About half the stomach was removed, the upper part of the opening sutured, and the lower part united to the jejunum through the transverse mesocolon in the usual way. The duodenal opening was closed. The patient left the nursing home on the fourteenth day, taking ordinary diet without pain or discomfort, and seven months later when last heard of he remained well.

THE SPECIMEN consists of a part of the duodenum, the pylorus, pyloric antrum, and a few inches of the body of the stomach, together with pyloric and a few omental glands. It was packed with gauze and hardened before being cut, so as to preserve its shape.

At the pylorus is a hard, nodular, infiltrating growth, sharply limited on the duodenal side, but encroaching a little on to the pyloric antrum. In the hardened specimen the stenosis is not so evident as it was clinically and at the operation, in spite of the fact that the whole specimen has shrunk to about two thirds of its original size. The tumour has ulcerated, but there is no fungous outgrowth into the lumen. It is of the infiltrating type like an epithelioma of the skin or tongue.

The polypoid tumours are entirely confined to the body of the stomach, there are none within three inches of the pylorus in the shrunken specimen. The large palpable one, originally about the size of a cherry, has shrunk very considerably, and many of the smaller ones, which originally appeared as sessile thickenings of the mucous membrane, can no longer be distinguished. There is no sign of ulceration or infiltration in any of the polypoid tumours, and from their absence in the pyloric antrum they would appear to have no connection with the carcinoma.

MICROSCOPIC EXAMINATION.—The pyloric tumour is a columnar celled carcinoma with areas of lymphatic infiltration in which the cells are polygonal, taking their shape from mutual pressure. The muscular coats of the stomach are involved, but the peritoneum appears to be intact over the growth. The polypoid tumour has the appearance of a localized overgrowth of the gastric mucous membrane and is everywhere separated by submucous tissue from the muscular coats. In its deeper parts the glandular tubules are fairly regular and normal in appearance, in the superficial parts they are very irregular and many small cysts are present. This layer forms the actual surface of the tumour, i.e., there is no separate covering of normal gastric mucous membrane. There is a slight fibrous core which appears to be continuous with the submucous coat. This core lies between and extends between the glandular tubules. The individual cells are of normal columnar type with basal nuclei, and the basement membrane is everywhere intact. The stroma is vascular, but there is no round-celled infiltration or other sign of inflammation. The mucous membrane between the tumours is normal. There is no evidence of chronic gastritis.

Diffuse polyposis of the stomach is evidently a rare disease, and, as most of the recorded cases have been found unexpectedly at post-mortem examinations, clinical records of the disease are still rarer. The older literature on the subject is confused by the inclusion of cases of single polypus, innocent or malignant, and of certain other gastric disorders.

The rarity of true polyposis is well brought out by some figures quoted by Myer⁸. In 7500 post-mortem examinations only 4 cases of gastric polypus were found (Obriuchow, Krinkenhuis). In several Russian hospitals the percentage of cases found post mortem varied from 0.007 to 0.04, and this included polypi of all kinds. Again, of 22 cases of gastric polypus collected from the literature by Ebstein⁹ only 3, or approximately one-seventh, could be described as cases of diffuse polyposis. Verse¹⁰ collected 55 cases of polyposis of the alimentary canal, of which only 4 were in the stomach. Further evidence is produced by Bilsfour¹¹ who recently published a case and stated that it was the first one observed in 8000 operations for diseases of the stomach at the Mayo Clinic.

The earliest case of which I can find a record was reported by Cruveilhier¹² in 1833. In this case forty scattered polypi were discovered at a post mortem examination, and he notes that they were attached to the mucous membrane, but moved freely on the muscular coats.

The first serious attempt to describe the disease was made by Menetrier in 1888. He gave a very minute and careful description of it and enumerated certain views on its origin which have been followed by many subsequent writers. He classified the cases into those in which there were scattered polypi over a large area of the stomach wall ('polyadenomes polypous') and those in which a large number of polypi were closely aggregated into a sort of plaque ('polyadenomes en nappe'). He further attempted to distinguish cases in which the overgrowth affected the deeper parts of the glands from those in which the ducts were mainly affected. Although his nomenclature suggests to the English ear a form of new growth he appears to have regarded the overgrowth of mucous membrane as inflammatory.

in origin, and he lays particular stress on the fact that the mucous membrane between the tumours always shows evidence of chronic inflammation. He is equally insistent on this point in the case of a stomach which contained one solitary polypus. Most of his cases showed signs of advanced arteriosclerosis, and he regards this as an important etiological factor. He reports 7 cases, but a careful consideration in the light of more recent knowledge compels me to exclude three of these from the category of polyposis. One was a single polypus, and one is described as having the mucous membrane thrown into longitudinal folds rather than polypus, while other evidence, such as intense hepatic cirrhosis, suggests that it was a case of chronic alcoholic gastritis. The third case that I exclude was almost certainly a 'leather-bottle stomach', the stomach is described

TABLE OF 19 CASES OF MULTIPLE

AUTHOR	DATE	SEX	AGE	MODE OF DIAGNOSIS	SYMPTOMS	EVIDENCE OF OTHER DISEASE
1 Cruveilhier ¹	1833	—	—	P M	—	—
2 Cruveilhier ¹	1833	—	—	P M	—	—
3 Richiardi ¹³	1846	M	51	P M	Diarrhoea	Chronic pleurisy
4 Brissaud ¹⁴	1885	M	79	P M	6 months loss of appetite and wasting	—
5 Leudet	—	—	—	P M	—	—
6 Menetrier	1888	F	62	P M	—	Phthisis. Tubercle ulcer in colon. Carcinoid in small int.
7 Menetrier	1888	F	40	P M	—	—
8 Menetrier	1888	F	52	P M	—	Cerebral haemorrhage
9 Menetrier	1888	M	35	P M	—	Left hemiplegia
10 Menetrier and Clunet ¹	1907	M	52	P M	—	Pulmonary tubercle
11 Menetrier and Clunet ¹⁵	1907	F	75	P M	No gastric symptoms	Atheroma
12 Menetrier and Clunet ¹⁵	1907	F	73	P M	No symptoms	—
13 Wegele ¹⁶	1909	F	59	Polyp found on gastric tube	—	—
14 Chosrojeff ¹	1912	M	36	Polyp in wash out	Haemorrhage. Loss of weight. Abdominal pain	—
15 Heimz ¹⁸	1912	F	35	Operation	—	—
16 Myer ⁸	1913	M	—	Polyp in wash out	20 years chronic gastritis	Syphilis
17 Von Saar ¹⁹	1918	F	56	Operation	—	—
18 Balfour ¹¹	1919	M	31	X ray and operation	3 years abdominal pain and loss of appetite	Had been treated for phthisis. Wassermann negative
19 MacPhedran ²⁰	1921	F	50	X ray	Pain after meals and constipation	—

MULTIPLE POLYPI OF THE STOMACH

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as being very small with all its coats enormously thickened, and there were secondary carcinomata in the liver

Including Menetrier's remaining 4 cases, I have been able to collect from the literature 19 cases only of multiple gastric polypi, i.e., cases in which there were at least two tumours. Single tumours and those of the very rare Brunnerian type, which are quite different, are excluded. Of these 19, however, 5 cases had less than six tumours present, so that only 14 can strictly be described as diffuse polyposis.

A brief table of the cases is appended. Many important facts are unfortunately missing, especially in the earlier cases.

OF THE STOMACH

MORBID ANATOMY	MICROSCOPIC APPEARANCE
red polypi Fixed in mucous membrane Free from muscles	—
red polypi but pyloric area free	—
red polypi the size of peas	—
11 polypi Areas round pylorus and cardiac orifice free No inflammation of mucous membrane	—
polypi mainly on greater curve Sessile and pedunculated	—
11 size of pea to hazel nut on lesser curve and posterior surface membrane appeared normal	Polyadenoma polypous Mucous membrane showed signs of chronic inflammation
As above	
11 red mass of polypi in plaque 8 cm x 4 cm and single isolated size of pea	Polyadenoma en nappe Mucous membrane showed signs of chronic inflammation
on lesser curve 4 cm from pylorus Forty polypi size of a pea mostly near carcinoma	Polypi as in Case 6 The growth on lesser curve was malignant
11 us polypi size of lentil to hazel nut and chiefly near the pylorus	As in previous cases, but with small mucous cysts
11 size of lentil to walnut near the lesser curve	Large polypi had fibrous core with mucous membrane of convoluted and cystic glands ten times the normal thickness over it. Small ones showed only the thickening of the mucous membrane.
11 carcinoma with single polyp near it	As large polypi above
11 surface studded with small soft polypi	Adenoma with core consisting of muscularis mucosa and fibrous tissue
11 c polypi size of pea to hen's egg	Polypi with carcinomatous metaplasia
11 on lesser curve Appeared benign	Adenoma " " becoming malignant
11 c polypi except near cardiac orifice and pylorus	—
11 on lesser curve with a single polyp some distance away	Adenoma No sign of chronic inflammation of mucous membrane
11 typical at the size of hazel nuts all within 5 inches of pylorus	Adenoma

In considering this table with my own case, certain points suggest themselves for discussion

Age—It has been often remarked that this disease mainly occurs in the aged, but this view clearly needs modification, for out of the 16 cases in which the age is known, 12 were not over sixty and 5 not over forty

Symptoms—The information on this head is very meagre, but it is evident from *Cases 11 and 12* that the disease may exist without giving rise to symptoms at all, and it is significant that the first 12 reported cases were all discovered post mortem. In other cases there were symptoms directing attention to the stomach, usually dyspepsia and hæmorrhage, and, in the treatment of a supposed chronic gastritis by lavage, the first ante-mortem diagnosis was made (*Case 13*), for a portion of tumour was found on the stomach tube. In the last two cases the diagnosis was made by radiography, which showed a characteristic mottling of the barium shadow. My own case shows, however, that this is not always evident. In *Case 18*, Balfour was fortunately able to excise the whole of the affected part of the stomach and this is, I believe, the first recorded instance of the deliberate diagnosis and radical treatment of the disease. Apart from radiography, the diagnostic point which Balfour chiefly stresses is the complete absence of free hydrochloric acid from the test meal.

Number and Position of the Tumours—Of 19 cases in which the stomach was examined either at operation or post mortem, in 5 there were less than six tumours, leaving only 14 which could be described as diffuse polyposis. In only a few of these was the precise situation of the polypi stated but in these the situation is rather striking. In 3 cases the tumours are described as being almost all near the pylorus, while in 4 others it is expressly stated that the pyloric area was free from tumours. The contrast is well brought out by a comparison of Balfour's case with my own, in his all the tumours were within five inches of the pylorus, in mine this was the only part of the stomach free from them. I can offer no explanation of this curious localization of the tumours. The greatest number of tumours counted in any one case was 250.

Association with Other Diseases—Menetrier regarded arteriosclerosis as a cause of this disease and found it in most of his cases. It is not uncommon in middle-aged people of the hospital class. There was evidence of tuberculosis in 4 cases and of syphilis in one. Syphilis has also been suggested as a cause, but in Balfour's case the Wassermann reaction was negative. I do not think there is enough evidence for any of these diseases to be regarded as the cause.

Association with Carcinoma—There were only 4 cases in the whole series of 20 (including my own) in which polypi were associated with carcinoma, and in each case the carcinoma was at or very near to the pylorus. The arrangement of the polypi differs in each case. In *Case 9* there were multiple polypi near the growth, in *Case 12* there was a single polypus near the growth, in *Case 17* a single polypus some distance from the growth, and in my own case there were multiple polypi all distant from the growth.

One's first thought on finding polypi associated with carcinoma is that they are secondary growths by permeation or implantation. On this view their peculiar arrangement in my own case is very difficult to explain and the tumours whatever they are, are certainly not in the least like secondary carcinomata. Alternatively, carcinoma may have started as a malignant transformation of one of a number of pre-existing polypi. P. Menetrier and Clunet in reporting *Case 12*, bring strong evidence in favour of this view. In this case there were two tumours on opposite walls of the stomach near the pylorus. One was a typical polypus and the other a larger sessile tumour adherent to the muscular coats. On section it proved to be early carcinoma. In my case, however, the carcinoma was of the flat epitheliomatous type and could hardly have originated from a polypus; moreover, the nearest part of the polypoid area was four inches away. In this case the carcinoma may have been a coincidence. One can say at any rate, that malignant change in gastric polypi is not very common, since in twelve post-mortem examinations at an average age of 58 years carcinoma was present in only two cases.

Nature of the Tumours—It is a striking fact that the work of Menetrier in 1888 still remains the most complete account of these tumours. His name, 'polyadenome', suggests

a new growth, but his description certainly gives the impression that he regarded the tumours as inflammatory in origin, and he is very insistent in each case on the presence of signs of chronic inflammation of the intervening mucous membrane. He is equally insistent on this point even in the case of a stomach which contained one single polypus and which is therefore excluded from the table above. Though I hesitate to disagree with so careful an observer, I am not convinced of this chronic inflammation. No other writer appears to have found it, and von Saar definitely states that in his case it was not present. In my own case Dr Lawrence Ball, who kindly examined the specimen for me, assures me that the mucous membrane between the tumours was perfectly normal and he examined it specially for signs of chronic inflammation.

As regards the tumours themselves, Menetrier's description still holds the field, though later knowledge has shown that the two types of glandular overgrowth which he attempted to distinguish, frequently co-exist in the same case. Of the later writers Wegle, von Saar, and Balfour describe the tumours as adenomata, while Chosrojeff describes his case as "polypus with carcinomatous metaplasia in part." The tumours are certainly not inflammatory in the sense of being infective granulomata. The glandular overgrowth is such that the mucous membrane becomes some ten times its normal thickness, and the tubules become irregular and frequently cystic. Although a certain degree of round-celled infiltration has been described by Menetrier, it is clear from his excellent pictures that the thickening is due to glandular overgrowth and not to inflammatory exudate. So far the tumour fairly justifies its title of adenoma. On the other hand, it has no capsule and not even a clearly defined margin, for its edge gradually merges into the normal mucous membrane in which respect it resembles an overgrowth due to chronic irritation.

One is familiar with the fact that overgrowth of squamous epithelium is produced by chronic irritation, and in certain cases by infection. The common wart on the skin is an example and though I believe no organism has been isolated its infectious nature is well recognized. There seems no reason why a similar infection should not produce overgrowth of the cells of a mucous membrane and, if so, such overgrowth would take much the form of these polypoid tumours. It would begin with a localized thickening of the mucous membrane (vesicle stage), and when this became big enough to drag on the stomach wall it would become polypoid. Finally, when of considerable size, it would drag down some of the submucous coat also, forming the fibrous core of the larger tumours so well described by P. Menetrier and Chinel.

In view of the absence of a capsule and the blending of the tumour with the normal mucous membrane I think these growths should be described as papillomata rather than as adenomata, and I am convinced that they will eventually prove to be something in the nature of infective warts on the mucous membrane of the stomach.

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THE APPLICATION OF THE WEDGE PRINCIPLE IN THE FASHIONING OF A TIBIAL 'BRIDGE' GRAFT

By R. E. KELLY, LIVERPOOL

THE weak point in all sliding bone grafts has always been their fixation. It has been generally conceded that a firm fixation gives the best results, although some authorities have stated that a too firm fixation is deleterious on account of the destruction of the bone-forming cells by excessive pressure. A moment's consideration of the actual pressure exerted by the muscles on the broken ends of any fracture ought to convince anyone that the pressure cannot be too great or the fixation too perfect.

If the surgeon uses a twin saw and cuts his graft with parallel sides, it is obvious that the bed is wider than the graft by the thickness of two saw-cuts. If he elects to bevel the sides of such an inlay, he certainly will get a closer union, but to ensure absolute contact and firm fixation, the bevelled graft must be fixed by pegs or sutures. Obviously, a better fit could be obtained by making the inlay exactly the width of the bed, but that would necessitate cutting the graft from the opposite limb. A 'bridge' graft, fashioned after the method of Hey Groves (the cricket ball graft), is difficult to make, requires careful fitting, and is exceedingly difficult to insert. The usual method of splitting the fractured bone in order to get the 'cricket ball' in place sometimes requires great force, and may even result in such splinting that the fixation becomes imperfect.

In a short paper in the *BRITISH JOURNAL OF SURGERY*, Vol. VII, No. 28, I described an operation for the relief of slipping peroneal tendons. Here a tiny graft of the fibula was cut with double-wedged sides. Mere pushing backwards of the graft fixed it firmly in its new position without the aid of a peg or suture. The application of this wedge principle may be used in any sliding graft. The inlay is fashioned in the form of a long wedge, so that, in moving it to its new position, it is gripped along its whole extent by the bed. The diagram (*Fig 171*) serves to illustrate the method. I also append x-ray photographs of a patient on whom I operated last year.

Mr X, age 34, was knocked down by a char-a-bone, sustaining a bad compound comminuted fracture of the lower ends of the left leg. He thinks that the wheel went right over his left leg. The wounds were excised, together with innumerable small contaminated fragments of the tibia. After a prolonged convalescence of a year, he had recovered,

with overlapping of the fibula and non-union of the tibia, the ends of this bone being about 1½ in apart. The destruction of so much tibia was accounted for by the loss at the first operation and the further subsequent removal of loose and necrosed fragments. At one time amputation was seriously considered, but as he retained a good blood- and nerve supply to the foot one refrained from the major operation in the hope of grafting

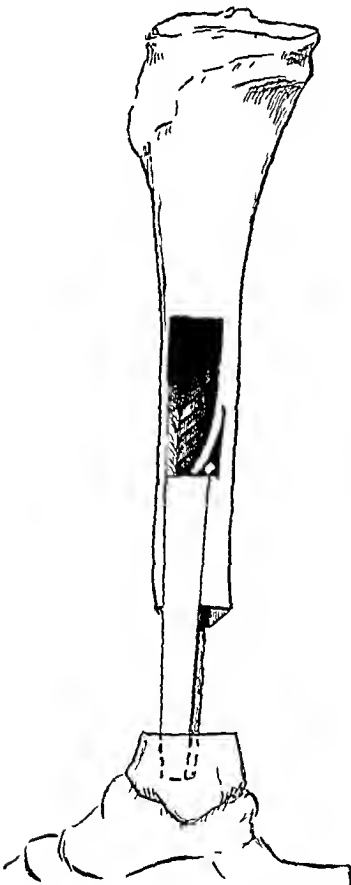


FIG 171.—Illustrating the method of applying the bridge graft

later on. The position at the time of grafting (October, 1921) was as follows. Overlapping of the fibula contact but no bony union, between the upper fibular fragment and the lower end of the tibia, free movement at the ankle and an intact foot, but quite useless for weight support (Fig 172). There was marked atrophy of the bones.

Operating with a tourniquet, a long incision was made over the internal surface of the tibia right down to bone, exposing almost the complete tibia. The periosteum was lifted with the flaps, and the whole operation carried out subperiosteally. If there is any virtue in the deeper layer of the periosteum from a bone-producing point of view, surely it is better to leave it *in situ* with its blood-supply intact, than to move it with the graft but sequestered completely from all vascular connections. I have so often seen the periosteum destroyed or torn off in many grafts despite special efforts made to preserve it that I am convinced it is safer to leave it attached to the tissues overlying it.



FIG 172—Before grafting



FIG 173—Immediately after grafting

When the whole internal surface of the tibia had been exposed, a wedge-shaped graft about 7 in long was cut with an Albee circular saw, in such a way that the upper end was about $\frac{7}{8}$ in wide, and the lower about $\frac{1}{2}$ in. In other words, the angle of wedge was about 1 in 20. Both saw-cuts were made at right angles to the internal surface of the bone. With a chisel the graft was freed from its bed and pushed down until its lower end impinged on and entered into a hole bored in the lower fragment of the tibia. This was done by tapping the top end of the graft using a hammer and chisel in the same way that a joiner works, the flat of the chisel placed on the top of the graft and the hammer used to tap the back of the chisel. The graft soon begins to bite into its bed, the amount of transposition being of course dependent on the width of the saw-cuts and the angle of the wedge. If one knows these exactly, the amount of translation of the graft before it is tight may be calculated with mathematical precision.

One must be taken that the wedge is not too blunt, otherwise the tibia may be

cracked before the required extension is attained. A further safeguard, however, to prevent the graft slipping upwards was made by placing a stop or catch at its upper end. A thin sliver of bone was cut with an Albee saw, about $\frac{1}{10}$ in wide and $1\frac{1}{2}$ in long upon the side of the bed above the graft. This was sawn through at its lower end, but left attached at its upper. A chisel was inserted into the saw cut, and the lower end of the slice levered inwards and fixed into its new position by the insertion of a tiny fragment of bone. This latch prevented any movement upwards of the wedge (see Fig 171). It could, of course, be done on both sides, but apparently this was not necessary. Fig 173 shows an x-ray photograph taken immediately after the operation. Note the atrophic character of the graft. The 'latch' does not show well, but it can be faintly seen on the fibular side of the tibia. Figs 174 and 175 were taken five and seven months later. The increase in strength and in thickness is well shown. Note also the



FIG 174—Five months after grafting



FIG 175—Seven months after grafting

development of buttresses. It is perhaps superfluous to add that all fibrous union, muscle etc., were removed from between the fractured ends before sliding the bridge graft into its new position. Further the rounded fibrous ends of the bone were sliced off.

I think this method of using a wedged graft to be of distinct value in cases of a similar nature where from atrophy of the bone, a surgeon is disinclined to use a graft about whose stability he is somewhat uncertain. Obviously it may also be used for a recent fracture, the only point to remember being that the fractured ends must be in perfect anatomical apposition, and kept so whilst the graft is being cut. The portion of graft which is not used, namely, that from the lower end, may be put back in the space left in the upper fragment. If the angle of the wedge is correct, the fixation is perfect.

Joiners often use a double wedge. Any graft, even a parallel-sided one may be sawn in two by cutting it diagonally. By sliding these wedges in opposite directions the graft may be wedged in its bed so firmly that extraneous fixation is not necessary.

CARCINOMA OF THE BONE-MARROW

By ALFRED PINFY, BIRMINGHAM

- I—INTRODUCTION
- II—ANATOMY OF THE BONE-MARROW
- III—BLOOD-VESSELS OF THE BONE-MARROW
- IV—LYMPHATIC CHANNELS OF THE BONE-MARROW
- V—DEVELOPMENT OF THE BLOOD FORMING TISSUES
- VI—CHARACTERS OF THE BLOOD PICTURE IN CASES OF CARCINOMA OF THE BONE-MARROW
- VII—MODE OF SPREAD OF CARCINOMA INTO BONE-MARROW
- VIII—EVIDENCES OF THE EMBRYONIC ORIGIN OF METASTASIS IN THE BONE-MARROW

I INTRODUCTION

THE existence of deposits of malignant tumours in the bone-marrow had been realized by morbid anatomists and surgeons a very considerable time before any definite explanation of the fact was available.

The early conception of the formation of deposits of cancer in the bone-marrow depended upon a belief in the existence of a 'cancerous diathesis', which was capable of manifesting itself in different regions of the body, either at the same time or successively. Sanson¹ described a case in which a woman with a scirrhus cancer of the breast of less than a year's duration, broke one femur while moving in bed. During the manipulations necessary for the reduction of this fracture, the other femur broke. At autopsy, there were many cancerous deposits throughout the skeleton, almost all the segments of the vertebral column were filled with tumour tissue, as were also the frontal bone and the medulla of each femur. In the last-named position, Sanson states that the tumour appeared to have grown from within the bone outwards. He sums up the case as follows: "qui a offert l'exemple peut-être le plus complet de ce qu'on nomme la diathèse cancéreuse".

The fundamental contributions to oncology which were made by Virchow made it necessary immediately to find some explanation of secondary cancerous deposits which did not depend upon the metaphysical conception of a 'cancerous diathesis'. It soon became obvious that the explanation of the process of formation of metastases was to be looked for in the blood or lymph. The conception of the formation of metastases by means of emboli consisting of cancer cells soon found abundant histological confirmation. The credit of pointing out the importance of a process other than embolism belongs to Sampson Handley. He has demonstrated that spread from a primary carcinoma of the breast is mainly by a process of 'lymphatic permeation' that is to say, growth takes place along the lymphatic channels from the primary focus outwards. In the course of such permeation, the epithelial cells may be destroyed by a process of perilymphatic fibrosis, or they may proceed directly along these channels until they reach another organ, where obstruction to their onward course will result in the formation of a metastatic nodule which is large enough to be detectable macroscopically. Obstruction to onward proliferation may take place at any part of the lymphatic system, and where this occurs there will be formation of a cancerous nodule e.g., in the skin.

The purpose of this paper is the detailed discussion of the evidence which relates to the problem of metastases in bone. In order to attack this much-disputed question, it is necessary to have a clear understanding of the anatomy of the bone-marrow, and a knowledge of the origin of this organ in the embryo. In a paper read before the British Medical Association at Glasgow in July, 1922, the present writer has described the macroscopic anatomy of the bone-marrow at different ages and only a brief summary of this subject can be given.

II THE ANATOMY OF THE BONE-MARROW

At birth all the bones of the skeleton except those of the cranium contain red marrow in which there is no fatty tissue either macroscopically or microscopically. As age advances fatty tissue appears in the marrow, but is not present in equal amounts in all the bones.

The vertebræ, sternum and ossa innominata contain red marrow throughout life, and only microscopic amounts of fat are detectable even in advanced age. The ribs are also storehouses of cellular marrow throughout life, but in advanced age a patch of fatty tissue usually appears at the anterior end of each rib and extends for about one inch from the costochondral junction.

The long bones present changes which are rather more difficult to describe accurately and briefly. At birth the limb bones contain red cellular marrow in the diaphyses as well as in the epiphyses. The cellular tissue is divided into compartments by firm bony trabeculae, and these take part in the series of changes which are normally associated with the attainment of the adult condition of the bone-marrow. Throughout childhood the marrow remains red, but fat is found in appreciable amount microscopically. It is not until the age of puberty is reached that macroscopically visible fat is found in the shaft. Thus first fat is visible just below the middle of the shaft in all the long bones and is surrounded by cellular marrow, which lies at the periphery of the medullary cavity. Extension of fatty change proceeds from this first formed mass of fat in both directions. The rate of spread in the distal direction is more rapid than in the proximal. Although the mode of fatty metamorphosis is the same in both the proximal and distal limb bones, there is one great difference, viz., the conversion into fat is complete more quickly in the distal bones. At the time when the whole of the radius, ulna, tibia and fibula are filled with fat, there is still a patch of red marrow at the upper end of each humerus and femur. This patch of red cellular marrow persists throughout life, microscopically it is obvious that there is a very considerable amount of fat even in this red patch.

The epiphyses of the long bones undergo a similar fatty metamorphosis, which is complete earlier than is the case in the shafts. The conversion of cellular marrow into fat is accompanied by disappearance of many of the bony trabeculae in the shaft, but there is less disturbance of these structures in the epiphyses. The residual patches of cellular marrow at the upper ends of the diaphyses are practically free from bony trabeculae.

The small bones of the hands and feet have not been examined sufficiently frequently to enable me to describe the changes in detail, but it is certain that conversion into fat is complete in them at an earlier age than is the case even in the radius and ulna, etc.

Summary of the Distribution of the Red Marrow in the Adult—In the adult the vertebræ, sternum, ossa innominata and the greater part of each rib contain red cellular marrow.

The only red marrow in the long bones of the limbs is found in a small area at the upper ends of the diaphyses. Longitudinal section of the long bones gives an incorrect picture of the exact distribution of the red marrow, whereas transverse section demonstrates that the fatty tissue is mainly confined to the axis of the medullary cavity, while the periphery still contains some cellular marrow for a varying distance below the lower edge of the definite mass of red marrow which is so well seen on longitudinal section.

Changes in the Distribution of the Red Marrow in the Adult—When one bears in mind the hematopoietic function of the bone marrow in post-natal life, as demonstrated by Bizzozero² and Neumann,³ it is obvious that any condition of the body which demands an increased supply of blood-cells, either red or white will throw a large amount of extra work on the hematopoietic depôts. If such a demand be comparatively slight or of short duration the existing tissue will suffice to produce the cells required; this is seen in the ordinary process of digestive leucocytosis. If, however, the demand is intense and long continued it will be necessary for the cellular marrow to hypertrophy in order to supply an adequate number of cells to the circulation. Such hypertrophy is seen in

leukemia pernicious anemia and many other conditions. It is therefore correct to state that the red marrow can increase in amount even in adult life in response to many varieties of stimuli.

III THE BLOOD-VESSELS OF THE BONE-MARROW

The gross anatomy of the blood-vessels of the bone-marrow is well known but apparently the extreme importance of a comprehension of the finer anatomy has escaped general notice. The best method of demonstrating the distribution of the blood-vessels in the marrow is by means of specimens which have been injected with a carmine-gelatin

mass, but even ordinary microscopic sections stained with eosin show the distribution of the blood channels quite distinctly. In the fatty marrow the blood channels are ordinarily well-formed vessels, but as soon as the red marrow is reached the conditions become much more difficult to follow. The red marrow is essentially a tissue consisting of innumerable blood channels with extremely thin walls. Outside these channels are hematopoietic cells. Fig 176 shows the arrangement of the blood-vessels in the red marrow. It is obvious that the great widening of the stream bed of the blood at the junction of the fatty with the red marrow must involve a great decrease in the rate

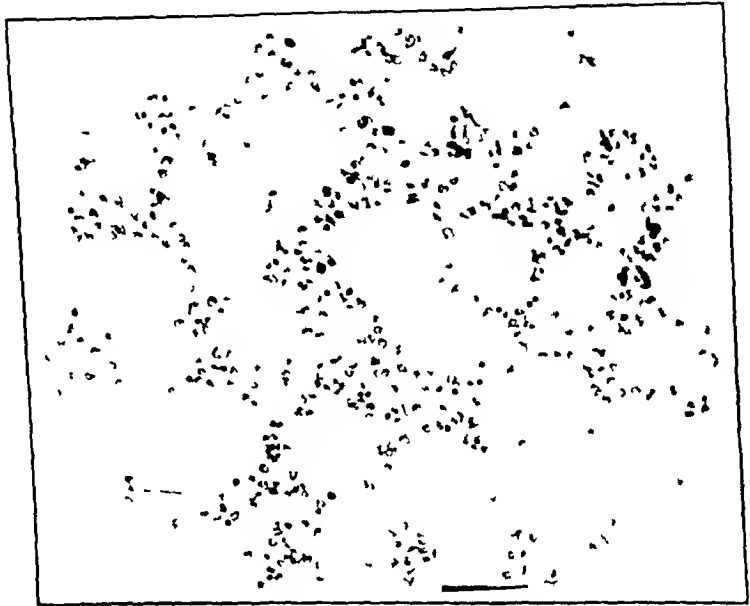


FIG 176.—The pale areas in this figure represent the blood channels of the marrow; the white areas correspond to fat while the dark elements are the marrow cells. This section was obtained from the marrow of the femur of a man, age 19. The extreme vascularity and the complicated arrangement of the course of the blood channels is well seen. ($\times 100$)

of flow in this area. It is important to recollect that the veins in the medullary cavity possess no valves although it would appear that those just outside the cavity possess more than the number usual in other vessels of the same size elsewhere. The anatomy of the vessels and lymphatics of the marrow was described in some detail by Schwalbe.⁴

IV LYMPHATIC CHANNELS IN THE BONE-MARROW

It has been frequently stated that the bone-marrow contains no lymphatic channels, but I have been unable to find any reference to experiments relating to this matter. Ziegler⁵ and Roger and Josue⁶ state that there are no lymphatics in the marrow, but give no reasons for making this statement. The extremely careful work of Sampson Handley on the subject of lymphatic permeation has made it imperative to attempt to settle the matter. Dewey and Noyes⁷ have used a very fine technique for the demonstration of lymphatic channels in teeth, and I have made use of this with slight modifications to make it applicable to such large objects as bones. The material used for injection was Prussian blue ground up with ether and turpentine in a mortar. I have found that it is possible to inject the lymphatic channels in the periosteum with comparatively little difficulty; the injection material passes from these channels into the bone and from it

into the endosteum, but I have been quite unable to find any evidence of a connection between these periosteal-endosteal lymphatics and the marrow tissue. If an injection is made with the modified Dewey Noyes cannula *directly into the marrow tissue* by screwing the cannula into the compact bone, the injection material passes into definite channels in the marrow tissue. The difficulty of regarding these as lymphatic channels was the fact that the injection material emerged from the veins at the large foramina.

The next step in the investigation of these channels was an attempt at a double injection, i.e., of both blood-vessels and lymphatics in the same specimen. The method adopted was as follows. The blood-vessels were injected with a carmine-gelatin mass, and when this mass had been completely solidified by cooling in iced water, an attempt was made to inject lymphatic channels through the Dewey Noyes cannula. Even if considerable force was employed in the attempt to fill lymphatic channels, the Prussian-blue ether-turpentine mixture could not be made to move along the marrow tissue. The necessary conclusion was that the channels, which were filled with the blue suspension in the previous experiment, were only blood-vessels. It appeared possible that a few lymphatics might enter the long bones along the tendons of attachment of the great muscles, but no communications could be detected between their fascial lymphatics and the marrow tissue.

In conclusion, the only statement possible at the present time is that modern methods of injection do not demonstrate the existence of any structures in the bone-marrow which resemble lymphatics in structure or distribution. The writer well realizes that negative evidence of this character is not necessarily of value, but the absence of lymphatic channels from the marrow is only a minor portion of the evidence upon which the present paper rests.

V DEVELOPMENT OF THE BLOOD-FORMING TISSUES

For the purposes of the present discussion there is little advantage in giving the well known details of the development of the blood in the embryo, but although no thorough consideration is necessary, it is essential to refer to a few of the finer details of the histogenesis of the blood-cells.

There appears to be little doubt that Weidenreich⁸ was perfectly correct in his contention that the white cells of the blood are not primarily true blood-cells but are really specialized developments of the primitive macrocytes, that is, cells formed in the primitive body cavity and only secondarily invariable components of the blood picture. When the development of the erythrocytes is considered, a very different

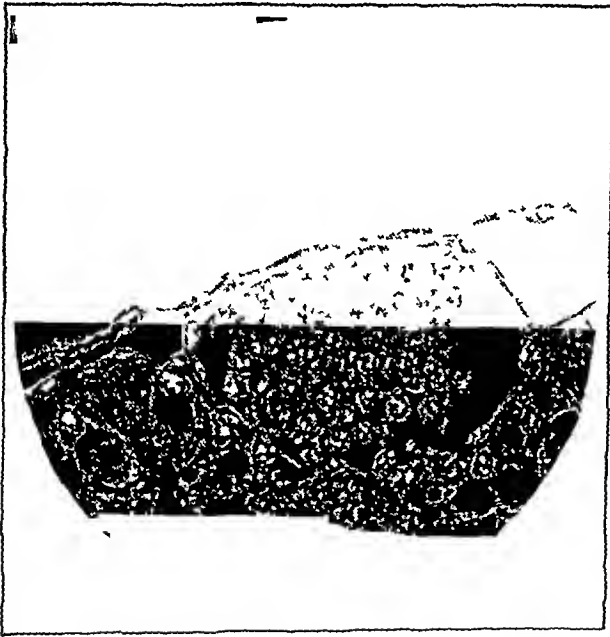


FIG. 177.—This figure shows one blood island from the wall of the yolk sac of the embryonic cavity. The clear portion in the upper part of the photograph represents the extra-embryonic cavity. The circumscribed cellular mass is the blood island which is surrounded by the primitive endothelium of the vessel while the red cells are in process of differentiation in the interior. The cells around the blood island belong to the yolk sac ($\times 100$).

arrangement is obvious. Figs 177 and 178 show blood islands on the wall of the yolk sac of the avian embryo and the human embryo respectively. It will be noted that the development of the red cells is an entirely intravascular process. Dantchkoff⁹ and

other observers have noted that the process of erythropoiesis in buds is purely intravascular, while leucopoiesis is extravascular.

The reason for this peculiar arrangement will be obvious from the description of the different mode of the origin of these two types of cells. The conception of two types of hematopoiesis does not involve the conception of two ancestral cells for the two types of blood-cells, the lining cells of the marrow produce red cells on the side towards the lumen, while white cells are produced on the other side. In this way an explanation of the ordinary hyperplasia of the red marrow is available. Under no conditions does one find a spread of the leucoblastic tissue without a contemporaneous spread of the erythroblastic. The converse is also true. Similarly, no injury to one process can leave the other completely intact. The present writer has dealt with this subject more fully in a paper read to the Pathological Society in July, 1922, with W. T. Hillier.



FIG. 17.—This figure shows similar appearances to those depicted in the previous one, but this specimen was obtained from a human embryo ($\times 150$).

VI THE CHARACTERS OF THE BLOOD PICTURE IN CASES OF CARCINOSIS OF THE BONE-MARROW

The realization of the hematopoietic function of the adult bone-marrow was soon followed by a partial comprehension of the grave alterations in the character of the blood picture which were observed to follow the presence of metastases in the marrow. The blood picture in such cases was found to resemble that of 'pernicious anemia' in many particulars, and in fact the first description of such a case by Ehrlich¹⁰ was that of pernicious anemia with incidental formation of a sarcoma.

The literature of hematology now contains details of many cases of this description, and it would appear that the alterations in the blood picture are independent of the site of the primary growth, and are only related to the presence of metastases in the marrow. Alterations of this 'pseudo-pernicious' type have been described in cases where the primary growth was in the stomach by Schleip,¹¹ Parmentier and Chabrol,¹² Harrington and Teacher,¹³ and Harrington and Kennedy,¹⁴ in cases of cancer of the breast by Epstein,¹⁵ Houston,¹⁶ and a case reported by G. R. Ward¹⁷ was almost certainly of the same character, although no confirmation was possible, as an autopsy was not permitted. Grunwitz¹⁸ reported a case of this type in which the primary growth appears to have been in the suprarenal gland, Reichmann¹⁹ described a case with similar alterations in the blood picture and a primary growth in the oesophagus, Schleip¹¹ gives details of such a case where the primary tumour was a colloid cancer of the appendix, and also reported another where the primary focus was in the jaw. There are doubtless other cases recorded in the literature, but I think that sufficient evidence has been presented to make it clear that there is no close relation between the site of the primary tumour and the character of the blood change.

It must be borne in mind that almost every case of cancer shows a definite degree of anæmia, but this is not of a specific character. Although the ordinary anæmia of cancer is not dependent upon the presence of metastases in the bone marrow, it is dependent upon changes in that organ. The chronic anæmia of protracted cases of cancer leads to an increase in the amount of red cellular marrow in the bones.

It would appear from the work of McMaster and his collaborators⁹ that hæmoglobin or one of its decomposition products is the essential stimulus to increased hæmatopoiesis after destruction of red corpuscles in the body. The cases of cancer in which the liver does not give a 'free iron' reaction are very rare, and are always those in which there is little or no increase in the amount of red marrow in the bones.

This increase in the amount of the red marrow is of great importance in connection with the present subject, as I propose to demonstrate that the red marrow is the site of deposition of cancer in bones. A brief description of the alterations in the character of the blood picture in these cases is necessary, because they are not usually quite easy to interpret, and also because some recent writers appear to be unaware of them and thus are led to publish misleading interpretations of a blood picture. In a recent controversy arising out of a paper by Izod Bennett and Dodds²¹ on the nature of the achlorhydria of pernicious anæmia, there has been an example of this misinterpretation. Dr A. E. Hurst²² suggested that the development of 'pernicious anæmia' in four cases of carcinoma of the stomach after total gastrectomy was evidence of the primary character of the achlorhydria of pernicious anæmia. Unfortunately, no detailed account of the blood picture was published, and therefore no definite statements can be made, but when one notes the great similarity between the condition of the blood in cases of carcinoma of the marrow and in pernicious anæmia, one would be chary of accepting the evidence of the true 'pernicious' character of these cases.

In this section I shall consider some of the cases of this type which have been published during the last fifteen years. One of the most carefully described cases with which I am acquainted is that published by Harrington and Teacher.¹³ The patient was a woman, age 64, who suffered from vague pains in different parts of the body, the most noticeable abnormality which was detected was a very definite anæmia, melæna was frequent. The authors report several blood-counts, of which the following is a typical example —

Red corpuscles	1,600,000 per c mm
Hæmoglobin	35 per cent
Colour index	1.09
Leucocytes	11,000 per c mm

A differential count of the leucocytes gave the following result —

Neutrophil polymorphonuclear cells	63.00 per cent
Eosinophil	0.70 "
Basophil	0.00 "
Lymphocytes	16.70 "
Large mononuclears and transitionals	18.80 "
Myelocytes	0.80 "

In counting 500 leucocytes they noted 29 megaloblasts and 4 normoblasts. The authors state that myeloblasts were present, but no figures are given.

Three weeks later the most marked change in the blood picture was a rise in the number of myelocytes up to 6 per cent of the total number of leucocytes. Polychromatophilia and punctate basophilia were well shown, but there was only slight poikilocytosis, while megilocytosis was very marked.

In this case the autopsy showed that they had been dealing with a case of scirrhus cancer of the stomach with numerous metastases in the ribs, vertebra and femur.

Harrington and Kennedy¹⁴ reported a similar case in which the total number of leucocytes per cubic millimetre was 10,000 and the primary tumour was in the stomach. The leucocytes were present in proportions somewhat similar to those in the first case, thus there were 2 per cent of myeloblasts while the myelocytes formed 0.5 per cent of the total number of leucocytes. The colour index was above 1.

Paumentier and Chabrol¹² reported a similar case in which the primary tumour was also in the stomach. Only one blood-count was performed just before death, and, although there was a very definite anaemia with a high colour index there were only 3500 leucocytes per cubic millimetre.

Schleip¹¹ has reported blood changes of this type in three cases, in which the primary tumours were in the stomach, appendix, and jaw respectively.

Houston's case¹⁶ of carcinoma of the breast with metastases in the bones showed a colour index of 1.28.

Reichmann's case of carcinoma of the oesophagus¹⁹ showed a colour index of only 0.66, but the blood picture was of the same character in other respects.

No good purpose would be served by referring in detail to the large number of other cases which can be found in the literature. It may perhaps be serviceable to give a summary of the changes of the constitution of the blood picture which can be regarded as sufficiently characteristic for the diagnosis of carcinoma of the bone marrow to be made during life.

CARCINOMA OF THE MARROW

'PRIMARY' ANAEMIA

- | | |
|---|---|
| 1 Reduction in the number of red corpuscles | 1 Similar reduction |
| 2 High colour index, not always above 1 | 2 Colour index usually above 1 |
| 3 Slight leucocytosis | 3 Usually slight leucopenia |
| 4 Leucocytosis due to increase of neutrophil polymorphs | 4 Relative lymphocytosis |
| 5 Anisocytosis, etc., well marked | 5 Similar appearances |
| 6 Nucleated red corpuscles present, both normoblasts and megaloblasts | 6 Always present, but vary in number at different times |
| 7 Myelocytes and myeloblasts present | 7 Myelocytes are not uncommon, myeloblasts are rare |

It will be obvious that the essential feature of the blood picture in these cases of carcinoma of the bone-marrow is the evidence of a grave disturbance of the erythropoietic organs while, in addition, there appears to be some interference of a stimulating nature acting on the leucopoietic mechanism.

At this point it might be instructive to refer to a different type of case which I had an opportunity of examining both before and after death.

The patient was a man, age 40, with well-developed Hodgkin's disease. Examination of his blood revealed the following surprising picture —

Red corpuscles	4,650,000 per c mm
Hæmoglobin	90 per cent
Leucocytes	45,000 per c mm

A differential count of the leucocytes revealed the following proportions —

Neutrophil polymorphonuclear cells	75 per cent
Neutrophil myelocytes	4 "
Myeloblasts	15 "
Lymphocytes	6 "

A second count a week later showed little change in the blood picture. In this case the blood shows no evidence of any interference in the process of erythropoiesis, but there is evidently much alteration in the mechanism of leucopoiesis.

I am able to find only two cases of secondary carcinoma of the marrow in the literature in which the blood picture was of this extraordinary character. The first case was that of Dieballa and Entz,²³ in which the leucocytes reached the surprising number of 112,600 per c mm and it is stated that there was no myeloid metaplasia in the liver and spleen. The second case was that of Bizarri,²⁴ in which there appears to have been a definite leukaemia of the myelogenous type with the well-known anatomical changes in the liver and spleen in addition to a cancer of the stomach. The second case is of little importance in the present discussion, but the first presents some difficulty. Dieballa and Lutz stated that both the liver and the spleen were enormously enlarged in their case,

and, as they offer no explanation of this phenomenon, it seems fair to leave their case out of consideration, and it has only been included in order to give completeness to the account of the varieties of blood change which have been known to be associated with cancer in bone-marrow

Blood changes of the 'pseudo-pernicious' type have aroused considerable interest, mainly from the point of view of diagnosis, but it appears to me that there is another and wider interest, as affording evidence of the mode of spread of cancer into the medulla of bones. In the section which deals with the development of the blood forming tissues, I have pointed out that erythropoiesis is an intravascular process, while leucopoiesis is extravascular. If the very equivocal case of Dieballe and Entz be disregarded, it will be noted that the essential change in the blood picture in cases of carcinosis of the marrow is a grave disorder of the distribution and appearances of the red corpuscles in the circulation, while the leucocytes show a far less intense degree of change.

It is usually admitted that lymphadenoma (Hodgkin's disease) is of the nature of a chronic granulomatosis rather than neoplastic in character. The mode of infection is quite unknown, and even the means by which deposits in foci distant from the primary granuloma develop is uncertain. There is no evidence pointing to a transfer of cells in these cases from one organ to another. Occasionally it is possible to see the lymphadenomatous process invading the walls of veins, but there is no evidence that cells capable of growth elsewhere are disseminated in this manner. Although the unknown virus of this disease may be carried in the blood-stream, there are no histological appearances which would lead one to suppose that the granulomatous tissue develops primarily in the blood vessels. As all the evidence shows that lymphadenoma is an extravascular process, it is not surprising that deposits of this granuloma in the bone-marrow lead to alterations

in the distribution of the circulating leucocytes. The case which is partly described above showed large masses of lymphadenomatous tissue in the marrow of many of the bones, and these were regarded as the cause of the strange blood picture which had been noted during life.

This case is, therefore, an example of the effect of an extra-vascular lesion in the bone marrow. It would appear perfectly justifiable to presume that the changes in the number and distribution of the red corpuscles in cases of carcinosis of the marrow are due to intravascular lesion.

At this point it must be admitted that cases of carcinosis of the marrow are on record in which there was no pseudo-pernicious blood picture. Middleton² has published such a case, where the primary growth was in the stomach. He remarked on the absence of reaction in the marrow

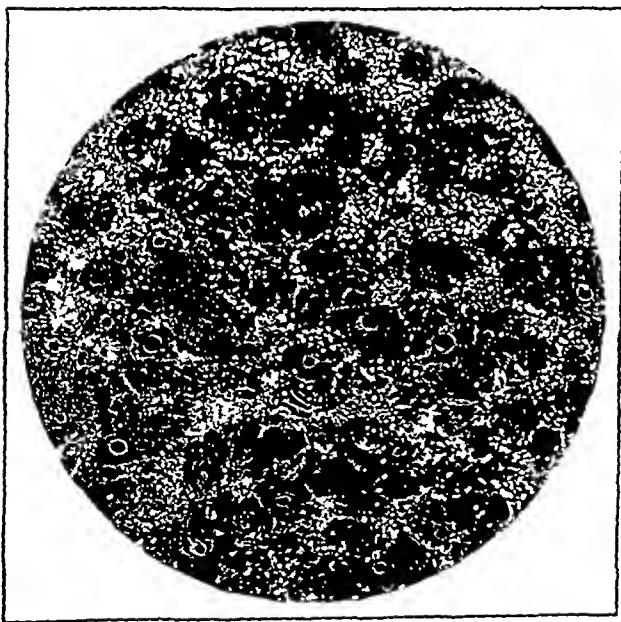


FIG. 179.—Case 4. This shows the appearance of advanced gelatinous degeneration of the marrow. It will be noted that in the degenerated areas there are no marrow cells and a lack of evidence of marrow reaction in a case of this description is not surprising. ($\times 100$)

tissue. Even if one cannot admit the existence of idiopathic cases of marrow aplasia in these malignant conditions, it is possible to conjecture as to the cause of the absence of the peculiar blood conditions. Fig. 179 shows a portion of bone-marrow from a case

of careinosis of the marrow (Case 4) in which no blood changes other than those of simple anæmia were detected during life. The photograph shows a very advanced stage of 'gelatinous' degeneration of the marrow. It would be surprising if so degenerate a tissue could show much sign of reaction to any form of lesion. If metastases of cancer settled in the bone-marrow at an early stage of the life-history of the primary growth, one would not expect that blood changes would be very well marked, because of the absence of hyperplasia, due to lack of previous anæmia.

A third cause of the absence of pseudo-pernicious changes would be great extension of the cancerous process, and extreme destruction of the marrow tissue. G. R. Ward¹⁷ made an interesting and important observation which bears a relation to this portion of my argument. In a case of aneurysm of the aorta which was pressing upon and eroding the vertebrae, the blood was found to present no deviation from normal. It appears certain that the effect of deposits of cancer in the bone-marrow is specific, and there are apparently insuperable difficulties about any explanation of the blood changes if the conception of an intravascular trauma is not accepted.

In all the cases in which pseudo-pernicious blood changes have been recorded in the literature, there has been a remark on the extremely dense fibrous character of the primary growth, i.e., these have been cases in which the disease has been present for a sufficiently long time to permit hyperplasia of the marrow to have taken place.

VII THE MODE OF SPREAD OF CARCINOMA INTO MARROW

It is not the purpose of this paper to deal with those cancers of bone which are due to extension from a primary growth directly into the neighbouring bones, only such tumours as are ordinarily regarded as metastatic will be considered.

The old conception of a 'cancerous diathesis' is not discussed in detail. The only modes of spread which fall to be considered are dissemination by the blood-stream and permeation of the lymphatics. Before dealing with the literature of the subject, I will give a brief account of the cases which I have had an opportunity of examining.

Case 1 — This patient was a woman, age 55, who was admitted to hospital with an ulcerated and discharging nipple. She stated that the condition was of two years' duration.

On examination, the left nipple was found to have been destroyed by ulceration, and a few hard glands could be palpated in the left axilla. The left breast was amputated and the axilla was cleared. Two months later the patient, who appeared to be well, was sent to a convalescent home, where she died within a month. The autopsy was performed on May 23, 1920, about twenty-four hours after death, and in abstract of the notes is given here.

The body was that of a stout woman showing early signs of wasting. There was a number of minute tumours on the pleurae, but the lungs appeared to be free from invasion. The liver, which weighed 2700 gm., was extensively invaded by metastatic deposits which appeared to bear a close relationship to the

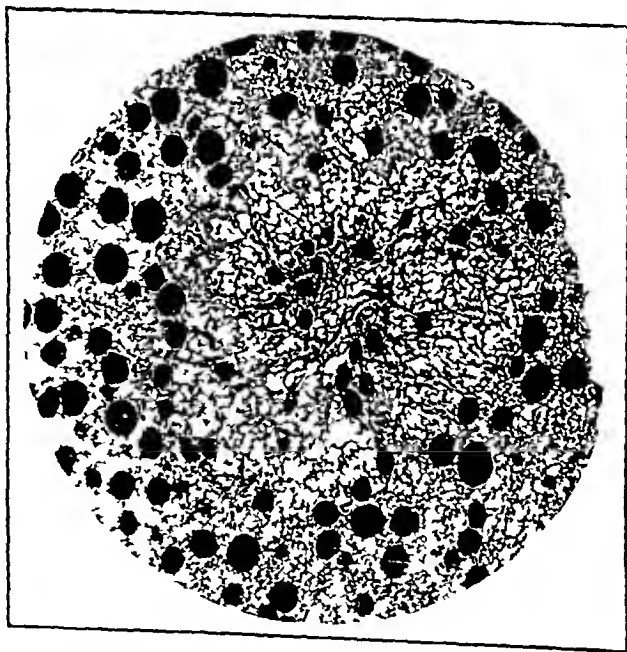


FIG 180 — *Case 1*. This figure shows the appearance of one of the smaller masses of metastatic tumour in the marrow of the femur. The fungus like mass of cells lies in the midst of hyperplastic cellular marrow and in the centre of the mass, the cells are seen to be arranged in a straight line ($\times 100$).

portal tracts. A few retroperitoneal glands showed signs of early invasion. The ribs were

extensively invaded by nodules of tumour, which, in places, filled the whole medullary cavity, but did not penetrate the bone, and the periosteum was not affected. The vertebrae were extensively invaded, and the metastases had produced softening in the affected bones.

The right femur was cut longitudinally and the cut section appeared red almost down to the lower end, but in this red tissue there were about a dozen white areas, the largest of these white areas were in the upper third of the medullary cavity, while the smallest were in the lowest part of the red tissue. The upper epiphysis contained red tissue and also tumour nodules, but the lower epiphysis was mainly fatty and was free from tumours. The bony trabeculae were almost undetectable in the red tissue of the shaft.

On microscopic examination, the primary growth was found to be a scirrhus carcinoma, and the deposits in the liver were of a similar character, although many of the nodules showed marked necrotic change. The metastases in the bones were of a more cellular character than the primary growth and, in the case of the smaller growths, resembled small radiating

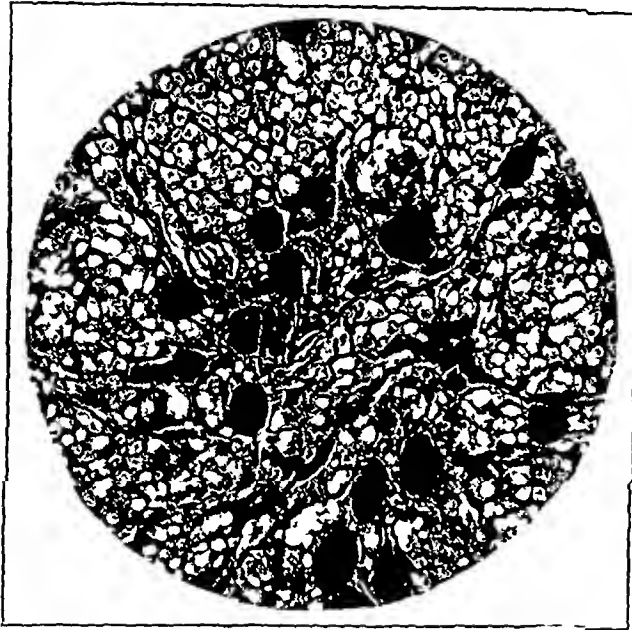


FIG 181—Case 1. This figure shows the central portion of the small tumour which is seen in the previous illustration. The row of cells in the centre is seen to be lying in a definite channel which is lined by endothelium. It will be noted that in neither of these figures is there any appearance of blood channels other than the one containing the cancer cells. ($\times 200$)

fungi in the surrounding hyperplastic marrow tissue (Fig 180). With higher magnification it was possible to see quite clearly that the tumour cells in the centre of such a mass lay in a definite channel, which was lined by endothelium, and there was no reason for supposing that this was not a blood vessel (Fig 181).

I have to thank Mr Seymour Barling for permission to refer to the above case, which was under his charge.

Case 2—The patient was a man, age 46 who was diagnosed on clinical evidence as suffering from carcinoma of a bronchus with secondary deposits in the liver.

The autopsy revealed the presence of a white mass of tumour at the root of the left lung, this invaded the lung tissue and appeared to arise from a bronchus. There were many metastases in the liver, which weighed 2500 gm. Both suprarenal glands were invaded by nodular tumour growth. The sixth rib on the left side was invaded by tumour which had perforated the periosteum on the pleural aspect, but had not invaded the pleura. The right femur

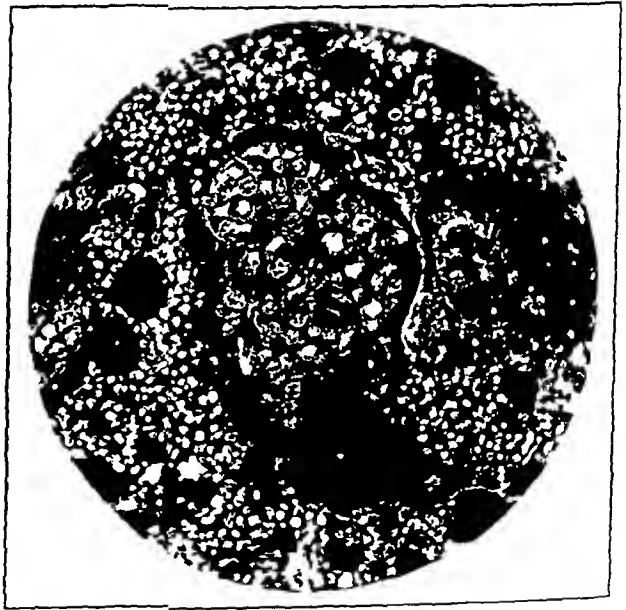


FIG 182—Case 2. This figure shows a small mass of metastatic tumour in the marrow of the humerus. The circumscribed mass is seen to lie in a definite channel and no blood vessels are seen in the neighbourhood. ($\times 100$)

showed a small mass of tumour tissue in the periosteum at the junction of the upper third with the lower two thirds of the bone, a needle could be pushed into this nodule for a distance of about an inch. On cutting the bone longitudinally, this small mass in the periosteum was found to correspond to a large white area of metastatic deposit in the medullary cavity. The red marrow had extended about half-way down the medullary cavity but no other metastases were found in it. The left humerus, on section showed the presence of a white nodule at the junction of the upper third with the lower two thirds of the bone, but no invasion of the periosteum could be found.

On microscopic examination, the bronchial tumour was found to be a cellular carcinoma of glandular type. The nodules in the bones were of similar structure, and it was easy to find plugs of tumour cells lying in channels which were lined by endothelium. The periosteal mass at the upper end of the femur did not show any signs of intravascular arrangement and was continuous with the tumour in the medullary cavity through the eroded compact bone. The appearances of the metastatic deposits in the humerus can be seen in Fig 182.

I have to thank Professor J. W. Russell for permission to refer to this case, which was under his charge.

Case 3—The patient was a man, age 55, admitted to hospital with an acute abdominal catastrophe. At operation a perforation of a stercoral ulcer in the cecum was found.

The autopsy showed that death was due to general peritonitis following perforation of a stercoral ulcer in the cecum in a man suffering from carcinoma recti.

No metastases could be found in any of the organs, including the bones. The only long bone which was examined was the right femur, and in this the red marrow was found to occupy the upper third of the medullary cavity, the remainder of that space being filled with fat.

On microscopic examination, the primary tumour in the rectum was found to be an adenocarcinoma. Sections from the red marrow at the upper end of the right femur showed the presence of emboli consisting of tumour cells in the blood-vessels. The tumour cells lay in vessels which had a definite endothelial lining and contained red blood corpuscles in a good state of preservation (Figs 183 and 184).

I have to thank Mr J. B. Leather for permission to refer to this case, which was under his charge.

Case 4—The patient was a woman, age 23, who had enjoyed good health until about six weeks before her death, when she noticed pain in the lower part of the back and the development of lumps in the neck. The blood was only examined on one occasion about four days before death and no marked deviation from the normal condition could be detected other than a slight decrease in the number of red corpuscles which were calculated at 4,000,000 per cubic millimetre.

The post mortem examination showed the presence of a tumour in the right lung. The upper lobe of this lung was white in colour and firm in consistency, being completely converted into tumour which invaded a bronchus. The lower lobes contained only a few discrete masses of tumour. Metastases were found in the bronchial glands, left suprarenal gland, and left ovary. There were thread-like lymphatics all over the surface of the heart, and these were found to be filled with tumour cells.

The bones were examined as far as was possible and extensive invasion was found. All the segments of the vertebral column were invaded in varying degrees, as were also most of the ribs. The sixth rib on the right side was almost fractured by growth but, on the inner aspect, the



FIG 183—*Case 3*. Shows the intravascular arrangement of the cancer cells in the marrow. In the vessel in the lower part of the field there are epithelial cells in the middle of the lumen while the periphery is occupied by red blood corpuscles. It will be noticed that there is practically no appearance of reaction on the part of the marrow tissue, and this is compatible with the fact that these metastases were of extremely early date in the history of this case. ($\times 100$)

compact bone still remained and separated the growth from the pleural periosteum. The tumour in this rib lay about one inch behind the costochondral junction.

The left clavicle showed a condition of almost complete fracture, but the posterior lamella of compact bone was not completely eroded. There were tumours in both humeri in each case these were situated at the junction of the upper third with the lower two thirds of the bone—the position of the red marrow normally present at this place in the adult. The red marrow in this case occupied an area which was no larger than that normal in persons of this age. The compact bone surrounding the medullary cavity was not eroded. In the head of the right humerus there was a nodule of tumour lying in the cancellous bone.

On transverse section of a bone at a point corresponding to a metastasis, it could be seen that the tumour mass lay completely in the marrow and did not invade the compact bone, although it lay in contact with it at the inner side of the cavity. The right femur was examined and showed a tumour lying in the red marrow at the upper part of the medullary cavity. No tumours could be found in the medullary cavity or epiphyses of the right tibia, which contained fatty tissue and no appreciable amount of cellular marrow. A nodule of cancer was present in the substance of the musculus tibiae anticus, and this was in contact with the tibia.



FIG. 181.—Case 3.

On microscopic examination the primary tumour in the lung was found to be a carcinoma, apparently derived from the epithelium of the alveoli and the metastases in other organs showed very great similarity in structure, this similarity was most marked in the case of deposits in the vertebral column (Fig. 185). The metastases in the long bones showed an alveolar arrangement but this was not quite so similar to the primary tumour as were the deposits in the vertebral column. The nodules in this case were more advanced than those in the previous cases, and it was not easy to find a plug of cells lying in a vessel but ultimately a mass of cells was found lying in a definite channel, which was lined by endothelium (Figs. 186 and 187).

The marrow tissue in this case was severely hyperplastic, but there was definite 'gelatinous degeneration' visible in some places (Fig. 179). This case illustrates the point that metastases in bone lie in the red cellular marrow and do not extend into the fat. There is no evidence in this case that the metastases had extended into the medullary cavity from the periosteum. The nodule which lay in the musculus tibiae anticus was only adherent to the tibial periosteum and no sign of invasion of the periosteal lymphatics could be found on microscopic examination. The tumour in the sixth rib shows the preference of metastatic deposits for the place at which fatty marrow passes over into the red cellular marrow.

I have to thank Professor J. W. Russell for permission to refer to this case which was under his charge.

These four cases are quite illustrative of the morbid anatomical features of carcinosis of the bone-marrow. The main points worthy of attention are —

1 The position of the cancer cells in channels which are lined by endothelium

2 The escape of the distal bones of the limbs

3 The absence of evidence of permeation of fascial lymphatics in the neighbourhood of the invaded bones

4 The position of the metastases, which is always in the red cellular marrow

5 The points of emergence of the tumour on the surface of the bones correspond to the places of exit of the veins

LITERATURE AND DISCUSSION

F von Recklinghausen²⁶ was the great exponent of the theory of the spread of metastases by the blood-stream into the marrow. His main contentions in favour of this conception were as follows —



FIG 15. — Case 4. Shows a portion of one of the many metastases in the vertebral column. The close resemblance of this secondary deposit to lung tissue is well seen. The masses in the vertebra were so advanced as to make it impossible to discover any arrangement in vessels ($\times 100$)

1 Metastases in bones occur in the interior of the medullary cavity, and only reach the periosteum by extension from this place

2 The masses in the subperiosteal tissue are always in the region of the large foramina which serve for the outward passage of the veins

3 The cancer cells in the marrow lie in definite channels which are arranged in a manner similar to that of the veins normally present in the marrow. He believed that these canals were blood channels for two reasons (a) Because no lymphatics were known to exist in the marrow, and (b) because no other blood channels than the invaded ones could be found in the affected areas. He admits that he was unable to discover any place in which such a channel contained both cancer cells and red blood-corpuscles

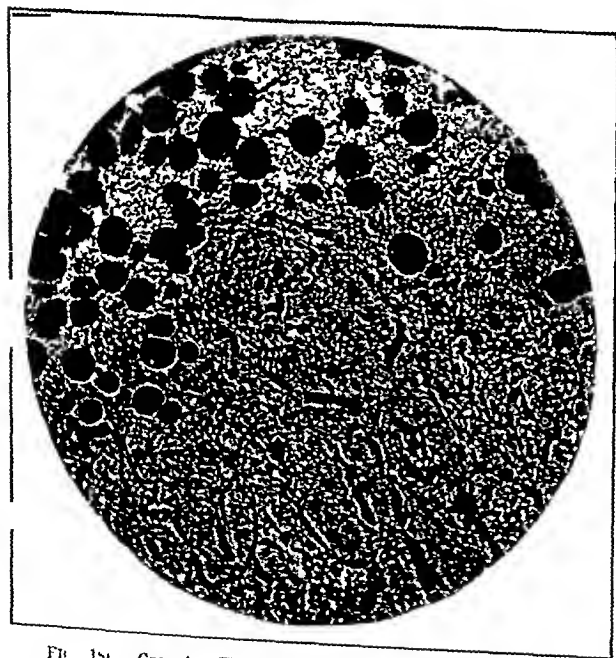


FIG 14. — Case 1. The upper portion of the figure shows rather hyaline marrow tissue while the lower two-thirds shows invasion by cancer. It will be noted that a mass of epithelial cells lies in a definite channel at the junction of the marrow tissue with the tumour mass ($\times 100$)

In the marrow of one of his cases Assmann²⁷ was able to find a capillary which was blocked by cancer cells, while another branch of the same vessel was quite free from invasion and only contained red blood-corpuscles.

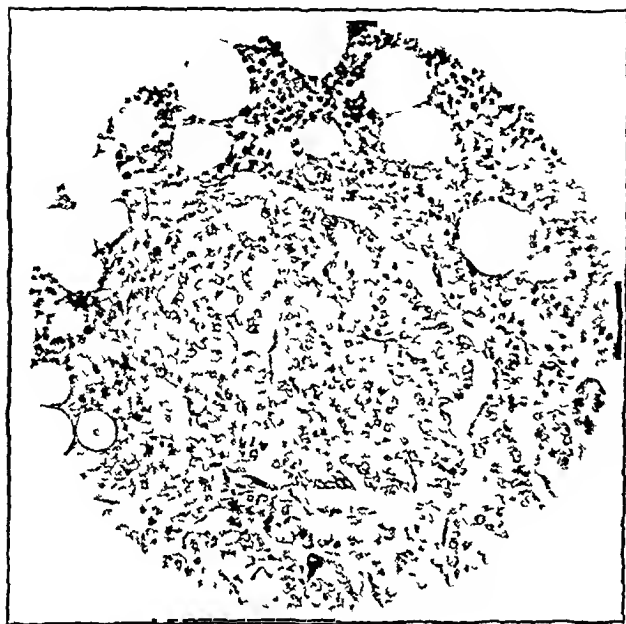


FIG 187.—Case 4. Shows the arrangement inside the channel which is seen in the previous photograph. An appearance of degeneration can be seen in the marrow tissue at the edge of the tumour mass ($\times 200$)

Erbsloh²⁸ was able to observe masses of epithelial cells in vessels, which still contained red corpuscles, in a case of carcinosis of the marrow secondary to carcinoma of the bile passages.

Goetsch²⁹ was inclined to believe that the subperiosteal nodules which occurred in some of his cases were earlier than those in the medullary cavity, but microscopic examination showed that the tumour in the marrow had undergone a greater degree of degeneration than had that in the periosteum, and was, therefore, probably the older. He was also able to observe that the cancer cells in the marrow lay in channels which occasionally were seen to contain red corpuscles.

It will be seen that the conception of dissemination by the blood-stream has appeared to

many observers to be the almost certain explanation of the metastases in the bone marrow, nevertheless, there is a school which holds that the invasion of bones is by means of lymphatic permeation. Sampson Handley, to whom the science of morbid anatomy owes its revival, is the great champion of the conception of lymphatic permeation as applied to the metastases in bones. His classical work on 'Cancer of the Breast'³⁰ has been the source from which I have obtained the details of the theory, which has done so much to advance the art of surgery in relation to the mammary gland, but, as far as it is applied to the invasion of bones, I am unable to accept it.

Many of Handley's statements are based on the most careful observation and are, therefore, quite immune from criticism, but the interpretations are liable to alteration. As far as I can gather the arguments from his work, they are as follows—

- 1 The freedom of the distal bones of the limbs from invasion by secondary cancer is regarded as incompatible with the conception of embolic spread, because these bones would be quite as liable to embolism as any others.

- 2 The liability of a bone to cancerous metastasis is said to increase with its proximity to the primary growth.

- 3 The femur is said to be invaded at the base of the great trochanter, but fracture usually occurs somewhat lower down on account of the thinner compact bone, invasion and fracture of the humerus are said to occur about the middle of the bone.

- 4 He disposes of the argument that deposits bear a close relation to the direction of the nutrient artery by pointing out that, in the humerus, the deposits are above the point of entrance of the nutrient vessel which is directed distally.

- 5 If the bones are invaded from the lymphatic plexus of the deep fascia, the point of attack should be the part of the bone which lies nearest to the cutaneous surface, and thus he says, is the case.

- 6 The escape of the distal bones is simply due to the fact that the patient usually

dies before the process of lymphatic permeation has proceeded sufficiently far to invade the deep fascia of the distal parts of the limbs

7 As additional evidence of the conclusion that bone metastases are associated with lymphatic permeation, he points out that the areas liable to cutaneous nodules and to bone metastases are similar in extent. He has demonstrated that cutaneous nodules are certainly due to permeation of fascial lymphatics

In a footnote he says, "I do not deny that in rare cases bone deposits may be the result of arterial or capillary embolism"

It has seemed to me that the most satisfactory mode of criticism would be the consideration of each of these headings separately, so that a final summary of the evidence in favour of the theory of cancerous embolism as an efficient cause of carcinosis of bones might be appended to this paper

1 The apparent immunity of the distal bones of the limbs is explicable on definite anatomical grounds. It will be recalled that, in the section of this paper which deals with the anatomy of the adult bone-marrow, it has been pointed out that the red marrow persists only in the upper ends of the shafts of the proximal bones of the limbs. It was also pointed out that the blood-supply of this cellular marrow was extremely complicated, inasmuch as the definite channels in the fatty tissue break up into a vascular system which is not unlike an angioma in arrangement. It is obvious that this widening of the stream-bed must be associated with a very considerable decrease in the rate of the blood flow. Decrease of the rate of blood flow is always associated with 'preventing' of the leucocytes, which are the solid bodies of the normal circulation. There appears to be no adequate reason for supposing that cancer cells would not be cast out to the periphery of the stream in a similar manner. In normal circumstances there is no evidence that leucocytes divide in the course of their journey in the main circulation, but in the red marrow they can often be seen lying in the periphery of the blood channels and showing evidence of division. These facts indicate that the stream at the periphery of the marrow vessels is slow, and that the development of cells in this situation would not be interfered with in a marked manner.

The process of embolism demands both the presence of insoluble particles in the circulation and also the possession of a suitable site of lodgement for such particles. The slowness of the circulation and the complication of the course of the marrow vessels appear quite adequate to supply the second factor.

All these points are a portion of the explanation of the fact that metastases in the bone marrow invariably lie in the cellular marrow and never in the fatty tissue.

2 If there is a greater liability to invasion on the part of the bones nearest to the primary growth, it may be due to direct invasion of the arterial system in the region, but my own observations and my investigations of the literature have not shown that there is any such predisposition.

3 Handley states that the femur is invaded at the base of the great trochanter but that spontaneous fractures occur rather lower down on account of the thinner layer of compact bone. The site of invasion corresponds to the area of red marrow in the normal adult femur. He states that the humerus is invaded about the middle of the shaft and also breaks in this position. He gives no reason for ignoring the possibility of invasion rather higher up, with subsequent spread in the distal direction. My cases show that the first invasion is at the upper end of the diaphysis and that dissemination can occur both upwards and downwards. Metastases in the medullary cavity are always situated in the red marrow, and growth along the cavity is associated with an increase in amount of the red marrow which always precedes the deposition of the metastases.

4 The arguments of Handley efficiently dispose of the idea that the direction of the nutrient artery is of any importance in this connection.

5 The position of metastases at the periphery of the medullary cavity is explicable on the basis of the embolic theory because of the lower level to which the cellular marrow reaches at the periphery. This is described in some detail in the section which deals with the anatomy of the organ.

6 The discussion of this contention was dealt with in paragraph 1 above

7 The resemblance in extent between the areas liable to cutaneous nodules and those liable to metastases in the bones is surely a very weak support for Handley's contention. Many cases with cutaneous nodules in an advanced state of development show no signs of invasion of the bones, and most certainly not all cases of carcinosis of the bones are accompanied by cutaneous nodules.

There are several other points which merit attention. Thus, there may be difficulty in the explanation of the spread of cancer cells in the circulation from the venous side to the arterial side without involvement of the lungs. M. B. Schnudt³¹ showed that small thrombi containing cancer cells were common in the capillaries of the lungs even in cases where there was no tumour in these organs on ordinary examination. Many of the epithelial cells in such thrombi appear to be destroyed, but he was able to observe that they might, on occasion, grow through the thrombus material, in this way cells may easily pass through the lesser circulation and be set free in the greater circulation and so pass to the bones.

If bones were invaded by permeation of lymphatic channels, it would be reasonable to suppose that examination of the periosteum would show evidence of invasion before the medulla contained any epithelial cells. In *Case 2*, where the periosteum of the femur was invaded, there was no evidence that the cells lay in any channels in this tissue, and in addition the periosteal tumour was in direct continuity with the cancerous mass in the medullary cavity. This bone is obviously one from which it is impossible to draw any definite conclusions, but the other bones in this case were found to contain tumour in the medullary cavity and not in the periosteum, and in their case it seemed impossible to conceive that invasion was from the periosteal lymphatics. In *Case 4*, the nodule which affected the periosteum of the tibia was a direct extension from the mass in the musculus tibialis anticus, and the lymphatic channels in the adjacent periosteum were unaffected.

In addition to the cases which I have had the opportunity of examining, there are many published cases which bear out my contention that the site of first settlement of metastases in bones is in the medullary cavity. In fact I have been unable to find any published cases, other than those of Sampson Handley, in which careful examination had led the author to any other conclusion.

VIII THE EVIDENCES OF THE EMBOLIC ORIGIN OF METASTASES IN THE MARROW

1 The absence of any histological or experimental evidence of the presence of lymphatic channels in the bone-marrow is an important argument in favour of the contention that carcinomatous metastases reach the bones by means of the blood stream. It is admitted that the proof of a negative proposition of this kind is always on an extremely uncertain basis of argument, but in the present case there are so many even more important evidences of the embolic theory that this one need not be unduly stressed.

2 The demonstration of plugs of epithelial cells in channels lined by endothelial cells and surrounded by red corpuscles in *Case 3* is evidence in favour of the embolic theory which is very difficult to confute. In other cases it has not been possible to demonstrate the intravascular position of the cancer cells with the same degree of certainty, but the presence of epithelial cells in passages lined by endothelium is regarded as weighty evidence that the position of the cancer cells is intravascular, although it has not been possible to show definitely that the passages are blood channels.

3 The objection to the embolic theory, which is based on the fact that the distal bones of the limbs are extremely rarely affected by secondary carcinoma, is dependent upon a deficient appreciation of the anatomy of the bone marrow. The embolic theory demands not only that the carcinoma cells should have access to the blood stream but that they should settle in a tissue and proliferate there. The settlement of an embolus demands the presence of certain anatomical factors, such as slowing of the blood stream

and a complication in the course of the vessels. These desiderata are found in the red bone marrow, although they are not present in the fatty marrow. The red bone-marrow is a place in which the stream-bed of the blood widens, the course of the vessels becomes more complicated, and the conditions for the lodgement of an embolus become correspondingly more favourable. Red marrow with its wide blood channels is absent from the distal bones of the limbs and also from the distal parts of the proximal bones and therefore these places are unfit for the settlement of emboli.

4 The site of the earliest metastatic tumours of bones is in the medullary cavity at the lower edge of the red marrow in the proximal bones of the limbs. Fracture does not occur until tumour tissue has spread along the shaft of the bone to a place where the compact bone is fairly thin. The spread of tumour tissue is preceded by a spread of red marrow. If there is hyperplasia of the red marrow in the bones, owing to previous anemia, the first deposition of metastases will not necessarily be at the upper end of the medullary cavity, but may be at the lower part of the hyperplastic red marrow.

5 The slowing of the blood-stream in the red marrow operates in a very definite manner: it results in the solid elements of the blood being sent to the periphery of the stream. This is seen frequently enough in the phenomenon of 'pavementing' of the leucocytes in the process of inflammation. Epithelial cells are certainly solid elements when compared with red corpuscles, and would, therefore, pass to the periphery of the blood-vessels of the marrow and proliferate there, where there would be little interference with their further development.

6 The grave changes in the composition of the blood picture are only explicable on the basis of an intravascular trauma to the marrow, as extravascular injury would result in changes in the distribution of the white cells of the blood. This latter type of change is well seen in cases of lymphadenoma affecting the bone-marrow.

7 The route by which emboli reach the arterial stream is not certain in all cases, but the work of M. B. Schmidt has demonstrated the frequency with which the vessels of the lung contain thrombi consisting, in part, of epithelial cells derived from a primary carcinomatous growth. These cells may be destroyed in the vessels of the lungs, but some of them may grow through the thrombus material and so give rise to emboli which are capable of colonizing elsewhere. This process would perhaps account for recurrences of carcinoma many years after removal of the primary growth. This explanation of the occurrence of late metastases is at least as plausible as the theory of uninterrupted permeation of lymphatic channels for a period of years.

8 The lack of observations on the involvement of the deep fascia in some of the cases of metastatic involvement of bones is another piece of evidence against the theory of lymphatic permeation.

9 The points at which carcinomatous metastases reach the surface of an affected bone correspond to the foramina through which the veins emerge.

IX CONCLUSIONS

The present investigation has led the writer to the conclusion that metastatic deposits in bones are due to arterial or capillary embolism. The main evidence which is brought forward is the detection of cancer cells in vascular channels in the bone-marrow. The vascular nature of these channels is demonstrated by the fact that they contain red corpuscles in addition to the epithelial cells. Evidence is also brought forward to show that the bone-marrow contains no lymphatic channels.

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SOME SURGICAL ASPECTS OF FILARIAL DISEASE

By F POWELL CONNOR, DSO Lt-Col, IMS, CALCUTTA

FILARIAL diseases in tropical and sub-tropical countries are responsible for a multitude of surgical conditions, ranging from trivial ulcers to surgical emergencies of the severest kind. When one considers that millions of people are infected with this nematode, about which we know so much in some respects and so little in others, one cannot but feel attracted towards the subject.

It is amply proved that filarial infection can exist without any signs or symptoms being exhibited by the victim. In some cases this infection can persist for a considerable period and cause no disability, while in others the earliest evidence of infection may be a serious lymphatic obstruction, inflammation, or other surgical complication.

F. bancrofti and *F. medinensis* are the two parasites responsible for the greatest amount of suffering as regards human beings, their definitive host. The life-history of *F. medinensis* is well known as are the inflammatory processes associated with the extrusion of the adult female from the tissues of the human host. But there is also a not uncommon class of surgical complications produced by this parasite to which I would like to refer, as sufficient attention has not been paid to them in the literature of the subject. These are the protean signs and symptoms which may arise from the irritative lesions directly due to calcified pieces of the dead worm remaining buried in the tissues. Acute or chronic cases of myositis, synovitis, inflammations of nerves, fibrous tumours or abscesses, and many kindred affections, may be met with, and offer considerable difficulties as regards diagnosis. Such sequelæ may not appear until months or years after the death of the worm, but the history of the eruption of other guinea-worms, or even the fact that the patient comes from a part of India where the worm is known to be common, should give rise to suspicion as to the real cause of the trouble.

The importance of realizing the surgical significance of the remains of the calcified guinea-worms in the tissues was only impressed upon me when acting as Consulting Surgeon to the Mesopotamian Expeditionary Force. Almost every Indian race was

represented in the Force, and I was given an opportunity of studying guinea-worm affections in patients coming from some of the most heavily-infected Indian provinces. A brief reference to a few selected cases will serve as illustrations.

A young Madrasí complained of pain in the scrotum. On examination a cord was felt about four inches long and rather thicker than the ordinary clinical thermometer, occupying the loose areolar tissue outside the left tunica vaginalis. It was hard, but not brittle, easily movable, and with two free ends. There was no tenderness. A round elastic mass, about $\frac{1}{2}$ in in diameter, could also be felt attached above and behind the left epididymus. A radiogram proved that these were calcified guinea-worm remains.

In this case, as in many, the calcified cord had a moniliform outline on the radiaplate. After removal by operation the structure of the cord resembled the roughly-drawn diagrams A and B, representing a cross section and longitudinal section respectively (Fig 188). The central axis (a) was fragmented, hard, and calcified and this was surrounded by a middle coat (b) of putty-like consistency and an outer coat of fibrous tissue (c). The print of this radiogram has faded too much to be worth reproducing.

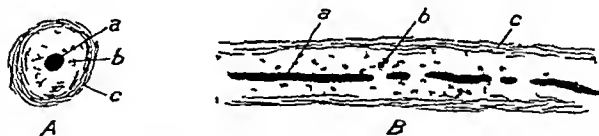


FIG 188.—Diagram to show the structure of the calcified cord. A cross section. B longitudinal section. a central axis, b the middle coat, c, the outer coat.

A store-keeper, age 35, was admitted into hospital complaining of a painful mass above the right heel. A hard, irregular tumour was felt in and around the tendo Achillis, flattened from before backwards, and with irregular edges projecting beyond the tendon laterally and anteriorly. The x-ray print (Fig 189) illustrates the condition admirably, except that the dense fibrous tissue surrounding the calcified cord is not shown.



FIG 189—Showing calcified guinea worm remains in right heel

As a general rule excision of the calcified cords is the proper treatment, but this can be very difficult, and in some instances the amount of disability may not justify an extensive dissection. Several patients were quite content to suffer a certain degree of pain or disability rather than undergo an operation.

Surgical affections connected with infestation by the *F. bancrofti* are very numerous. They may be broadly classified as being either inflammatory in nature or due to lymphatic obstruction, and quite often these two types are combined. Some of the well-known complications and sequelae are—

1 Filarial fever, which is often associated with elephantiasis, cellulitis, orchitis, etc. The only evidence of infection in other cases is the presence of microfilariae in the blood. Erysipelatoid attacks may also occur at irregular intervals.

2 Abscess and gangrene, most commonly serotal.

3 Orchitis, acute hydrocele, acute arthritis or synovitis.

4 Lymphatic varices, fistulae, gland varices, lymph scrotum.

This patient had been infected with guinea-worm in Jodhpur State during three years' residence there, and stated that twenty-one worms had been extruded from his legs, all below the level of the knees.

Other instances of this interesting surgical condition are shown in the x-ray prints (Figs 190, 191, 192, 193). The diagnoses made in these cases were, respectively, chronic rheumatism of the ankle joint, chronic traumatic synovitis of the knee joint, periostitis and sciatica. This proves how very baffling these conditions may be, and how ineffectual the treatment is until their true nature is ascertained.



FIG 190—Calcified guinea worm infestation diagnosed as chronic rheumatism of the ankle-joint

- 5 Chyluria and chylous effusions into the peritoneum, tunica vaginalis, etc
- 6 Elephantiasis of the scrotum, legs, arms, mammae, vulvæ, and skin

These complications vary much in severity, and though in rare instances they may rapidly prove fatal, in the great majority of cases the effects are transient, though liable to recur at decreasing intervals and with increasing severity



FIG 191 — Another case diagnosed as chronic traumatic synovitis of the knee joint

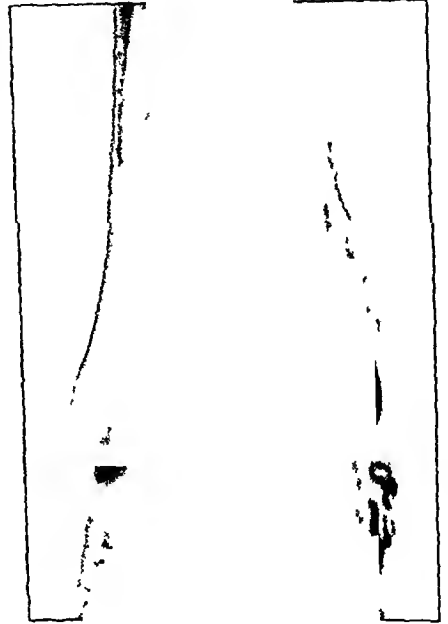


FIG 192 — Another similar case mistaken for periostitis

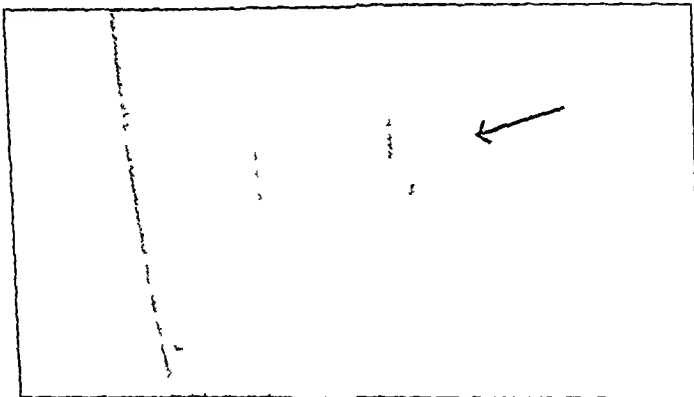


FIG 193 — A case of calcified guinea worm remains diagnosed as scrofula

It will not be necessary to describe even the more important of these surgical affections, as they are all well known and fully described in text-books on the subject

There are many problems connected with filariasis which still await solution. We cannot account for the age incidence of the various complications, because we do not know how long it takes for the parent worm to develop in the tissues and produce free

embryos Surgical complications scarcely begin to appear before the age of 10 years, they are most common in years 20-40, and after 50 years become quite uncommon

We do not know why hyperfilariasis does not occur in more patients continually exposed to the bites of infected mosquitoes, and though it appears that all classes are liable to infection why is it that some individuals escape altogether? The wife may be infected and the husband escape. Women appear less liable to infection than men, and perhaps the poorer classes are more infected than those who are better off

Probably gravity has a good deal to do with the common infection of such parts as the scrotum, the external genitals, legs, etc. These parts also contain large numbers of lymphatics. Whether the warm and moist surfaces in these regions help filarial growth we cannot say

Elephantoid enlargements are the most obvious surgical complications which we believe to be associated with filarial diseases. But there is no certain chain of evidence to prove to us how these hypertrophies occur. They are only found in regions where filariasis is common, and there is much evidence to prove that filarial infection is the necessary link in the etiological chain. But I am not at all satisfied that we know the exact pathology. Is it the microfilariae which are responsible? We know them to be apparently innocuous in the great majority of cases when found in the circulating blood, but it may be otherwise if they are shut off in a confined space by blocked lymphatics. Under such conditions the restless movements of their teeming millions may well produce irritative effects on the endothelial walls. Though adult filariae may block large lymphatics and lymph glands, it does not seem likely that they can produce such extensive lymph stasis as occurs in these cases. Their living bodies or calcified remains are not found in sufficient numbers to account for the oedema and fibrosis which result. One must remember, however, that in the case of such minute nematodes, absorption and disintegration of their tissues would occur in a very short space of time.

We are forced to conjecture that elephantoid thickenings are the result of either the irritative or toxic effects produced by the worms or ova, or that some concomitant infection such as a streptococcal invasion is responsible. We have ample evidence that such streptococcal invasions do occur, in that erysipelatoid inflammations commonly complicate the cases, and streptococci can be readily obtained by puncturing lymph spaces or lymphatic glands. These attacks exhibit an extraordinary periodicity which it is not easy to account for, unless it be that the intervals between the attacks represent the period of short immunity produced by each exacerbation.

There are several other points which are of interest in connection with this secondary streptococcal infection. It was found during the war that tissues infected by streptococci—generally introduced with multiple minute foreign bodies, such as fragments of missiles—were liable to a very serious inflammatory reaction if again interfered with, even when weeks or months had passed after the original injury. This is not true in the case of the erysipelatoid inflammations affecting elephantoid enlargements. Surgical operations can be performed with impunity on filarial tumours a short time after an attack of inflammation has subsided. It is true that suppuration is not unknown in such cases, but it is quite uncommon. It is even possible to implant gross foreign bodies, such as large strands of silk in the operation of lymphangioplasty introduced by Sampson Handley without necessarily reproducing a streptococcal cellulitis. It therefore seems obvious that the streptococci met with in these cases are not of an intensely virulent type.

It is a curious fact that in the case of the calcareous threads left behind quite often after the death of adult guinea-worms, and generally broken up in a moniliform manner, we seldom meet with a similar streptococcal invasion. In these cases the foreign body is a large one, and one would expect a greater degree of tissue irritation.

One must admit that the pathology of these erysipelatoid inflammations in connection with filarial enlargements is not at all well understood. Given a streptococcal infection the solid oedema and fibrous thickening of the subcutaneous tissue is easily explained. It is exactly what is met with after many attacks of cellulocutaneous erysipelas in non-filarial cases. An infant under my care a short time ago had had several attacks of cellulitis of

this type, resulting in a brawny œdema of the legs. It is now slowly disappearing, but one can readily imagine that consecutive attacks at regular intervals would cause a good deal of fibrosis and produce a condition of elephantiasis not differing in any essential respects from the cases generally described as filarial.

A very acute condition described as 'septic phlebitis of the spermatic cord' by the late Colonel R. Bird, I.M.S., and also known as 'funiculitis' (Castellani), occurs in India. This is a very dangerous form of streptococcal invasion of the spermatic cord rapidly spreading upwards and downwards and causing streptococcal septicæmia, if not promptly dealt with by surgical methods. I have often wondered whether some of these cases are initiated by filarial infections. The terribly acute nature of this streptococcal invasion is, however, rather in contrast to less severe types met with commonly in filariasis. But the wholesale lymph stasis in the latter, which is absent in funiculitis, may to some extent explain this. I would be glad to know whether any experiments have been carried out to ascertain the degree of virulence of the strain of streptococci commonly found in filarial infections.

The etiology of hydrocele in India has been very little worked out. Undoubtedly some of these cases are filarial, but in a great many patients no evidence of this infection exists. Specimens of fluid from ten cases were examined recently, none of these produced any growth on culture, two showed numerous filarial embryos, one numerous spermatozoa, while in most cases tyrosin and cholesterol crystals—particularly the former—were abundant. In many instances the cord presents no evidence of thickening or disease, but small patches of subacute inflammation can be found on the epididymis. No further elucidation, however, has so far been found to account for these patches. Of the many methods of cure practised in India for hydrocele, open operation is the only one which can be adopted as a radical cure. Various modifications are carried out by surgeons, but it may be fairly stated that they all attempt to achieve the desired result either by removal of the secreting layer of the parietal tunica vaginalis or by its eversion or plication. It would be of great labour-saving value if, by some non-irritating chemical or bacterial agency, the endothelial lining of the tunica vaginalis could be obliterated without causing injury to the testis or neighbouring tissues. It is also possible that a permanent filtration channel could be devised, on the lines attempted in ascites, by implanting a foreign body or a piece of fascial tissue in a window created for the purpose in the parietal wall of the tunica vaginalis. The open operation is a very satisfactory one, but in districts like Bengal and Orissa, where a large percentage of the population is affected in this way, a simpler procedure is badly needed.

Great ingenuity has been exercised by surgeons in devising operations for elephantoid enlargements. A very brief reference to some of them will be made here. Generally speaking, the surgery of pendulous thickenings is very successful, but similar affections of the extremities are much more difficult to deal with.

In the lower extremity, decortication of the whole affected area followed by skin-grafting and 'lymphangioplasty' (Simpson Handley), is not a very satisfactory procedure. The removal of considerable strips of tissue, muscle-deep from the lowest part of the swelling to a region of healthy tissue above (Kondoleon), has been found to be the most successful operation.

The operation for the removal of elephantoid enlargements of the scrotum and sheath of the penis is one which has developed a good deal within recent years. Considerable variations exist in the type of operation performed in various parts of the tropics. The following points sufficiently indicate the procedure followed by me—

- 1 The incision varies with the size and variety of tumour, and is not very important except that the perineal flaps should be made as wide as the healthy tissues will allow of. In very large tumours it is wise to isolate and lay bare the penis and testicles before fashioning the perineal flaps. In the case of smaller tumours it is quicker to cut these flaps and expose the testicles from behind and deal with the penile sheath last.

- 2 Much time is saved by tearing through the tissues with gloved fingers as soon as the soft œdematous layers are reached.

3 Blood-vessels, which are generally of considerable size, should be tied with catgut after clamping, twisting is not a safe procedure

4 The testicles should be accommodated beneath the perineal flaps, when these are sufficiently large to cover them. Failing this, they can be placed more easily in pockets excavated by the gloved fingers in the subcutaneous tissues of the adjacent parts of Scarpa's triangle

5 Drainage is not generally necessary

6 It is important to fix the fibrous sheath of the penis at its base by catgut sutures to the adjacent skin edges, to prevent retraction. I never utilize the preputial mucous membrane, though often tempted to do so, to cover the distal portion of the raw surface of the penis. It is very liable to solid œdema. Skin-grafting can be done at once or after a week by Thiersch's method

7 The efficient dressing of these cases is most important, and the method introduced by the late Colonel C. R. Stevens, I.M.S., is very suitable. It is by means of rolls of 1-inch lint soaked in 1 per cent picro lotion or normal saline. About four inches of the beginning and end of each roll are applied in turn to the surface of the belly, perineum, or inguinal region while the central parts of the bandages are wound round the penis. These tails are then held down by an ordinary double spica bandage after the usual dressing of gauze and cotton-wool has been applied. The lint becomes sufficiently stiff on drying to keep the penis comfortably cradled.

8 Every precaution must be taken to prevent any soiling of the wound by urine

The only weak part of this operation is the Thiersch's skin-graft of the penis. No suitable flap or modification of the Indiran operation (as for rhinoplasty) has yet been devised to replace it. Quite recently I have tried a new device and have been astonished at the success obtained. This consists in cutting a sufficiently large flap from the thick œdematous tissue covering the region of the dorsum of the penis. This is pinned down at the end of the operation with a razor and curved scissors till it is barely thicker than the normal skin of the penis and is then used to cover up that organ completely. Contrary to expectation, this skin has become quite soft and pliable after a few days, and if this result is always obtained, this procedure will remove the only real defect of the operation. It would seem that the skin and subcutaneous tissues of the dorsum of the penis and of the pubes are quite capable of filtering off their own lymph if not embarrassed by the lymph stasis of the scrotum and neighbouring parts.

UNUNITED FRACTURES DUE TO WAR INJURIES: WITH END-RESULTS OF OPERATIVE TREATMENT IN 100 CASES.

By A. PHILIP MITCHELL, EDINBURGH

As a result of the frequency of ununited fractures in gunshot injuries of the extremities, the operation of bone grafting acquired greater importance and has required to be extensively practised. In this paper it is not proposed to discuss the relative merits of the operations in use and experimental work that has been done in regard to the fate of bone-grafts, but to record the results of the experience gained from a personal study of 77 patients in the Military Orthopædic Hospital at Bangour during a period of three years ending March 31, 1921, and also of 23 cases during a period of eighteen months in Craigleith Ministry of Pensions Hospital, to describe the operations which the writer was led to adopt, and, by illustrative cases, to bring forward for consideration some of the important points in connection with the pre- and post-operative treatment.

The question of the fate of bone grafts when in process of conversion into normal bone is reserved for a subsequent communication in which the results of an experimental investigation, not yet complete, will be fully discussed.

GENERAL CONSIDERATIONS

In 61 cases the operation carried out by the author has been a primary one aiming at bone replacement, and in 24 cases unsuccessful attempts had been previously made by other surgeons.

It is important to note that the hundred cases for this study are unselected. The gap between the ends varies from 1 to 12 cm. and is filled with fibrous tissue. The fragments are frequently tapering, brittle and sclerosed, and the medullary canal is closed. When this osteosclerosis is extreme, it extends for two or three inches along each fragment, the periosteum being replaced by fibrous tissue. The surrounding soft tissues are also fibrosed and adherent to the bone. As such tissues bleed freely, and the bleeding is difficult to arrest—thus being a not uncommon occurrence in cases of the tibia and humerus—a complete excision of all fibrous tissue is advisable. The presence of lurking bacteria with a hæmatoma might result in failure of the operative treatment.

The importance of general causes of non-union of fractures is largely academic, and it is to be emphasized that local causes play a vastly greater rôle. The causes acting locally in the cases in the present series were as follows—

1 Primary loss of substance	55 cases
2 Displacement	12 "
3 Sclerosis and latent sepsis	5 "
4 Sclerosis with plating and wiring	4 "
5 Sclerosis and gap	12 "
6 Sclerosis	12 ,

The publication of the results has been delayed so that a sufficient period might elapse for most of the patients to resume civil employment and test the strength and utility of the reconstructed limb. Since the majority of the patients are pensioners from Edinburgh and neighbouring counties, it has been possible to keep them under observation from the time of operation up to the present date, and to make frequent radiographic examinations.

Table I—ANALYSIS OF CASES

BONE	NUMBER OF CASES	UNION BY OPERATION	FAILURE	PARTIAL SUCCESS	UNION BY CONSERVATIVE TREATMENT	NO TREATMENT REQUIRED OWING TO SLIGHT DISABILITY
Ulna	Left = 26 Right = 10	30	0	0	0	6
Radius	Left = 18 Right = 8	20	1	1	3	1
Humerus	Left = 6 Right = 13	16	1	2	0	0
Tibia	Left = 4 Right = 11	10	1	0	4	0
Femur	Left = 2 Right = 1	1	0	0	2	0
Fibula	Right = 1	0	0	0	0	1
	Total 100	77	3	3	9	8

PRE-OPERATIVE TREATMENT

The operation result and ultimate function of the limb are influenced to some extent by the nature of the pre-operative treatment that has been employed. Whilst the bone lesion may be the chief cause of the resultant disability, other tissues have frequently suffered considerable damage with consequent loss of function. A prolonged sepsis of the gunshot wound has frequently resulted in considerable destruction of muscle tissue and loss of function—marked limitation of pronation and supination, stiff fingers, and maybe loss of movements at the wrist, elbow, or shoulder joint, and lastly, an important nerve may have been severed or partially destroyed. All cases should have the benefit of hydrotherapy, massage, and active and passive exercises.

As regards deviation of the hand resulting from non-union in the lower third of the radius or ulna, very little can be done at a late stage by pre-operative treatment, but in the early cases this deformity can be avoided by means of a short plaster of Paris splint. This pre-operative period therefore need not be wasted, as non-union is seldom the only thing wrong with the limb.

OPERATIVE TREATMENT

As it is impossible to state after what period gunshot wounds are quite free from the danger of latent sepsis, my practice has been never to proceed to the bone graft operation until the wound has been soundly healed for at least twelve months. Referring to the tabulated data of the cases, it is shown that in most instances the original wounds had been healed fifteen months or longer before the patients came under my care for the appropriate reconstruction operation. Further, it is interesting that latent infection was first encountered in 6 cases all operated on within the last year.

Since there is no sure means of determining whether or not latent infection exists, an operation in two stages has been carried out by some surgeons. Such a procedure should certainly be adopted where prolonged sepsis of the original wound has resulted in extensive scarring of all tissues at the site of non-union. At the preliminary operation all sclerosed tissue is completely excised. The wound is then closed and a period of fourteen days allowed to elapse before proceeding to the grafting operation. Should a flare-up occur during this period, the infection can be much more easily controlled than if the complete operation had been carried out. But infection and a successful graft are

not incompatible. Case 17 illustrates this ability of a graft to thrive despite severe infection of the surrounding tissues. Figs 194 and 195 show the condition present before, and five months after, operation. Nevertheless, of the attributable causes of failure in bone grafting, it must be admitted that, above all other causes, sepsis is the great bane of this operation.

The key-note of surgery in ununited fractures should be absolute simplicity. The most perfect carpentry will not be followed by osseous union strong enough to restore satisfactory function if in carrying out the graft operation, the following anatomical, pathological, and general technical principles have received insufficient attention —

- 1 The importance of making the skin incision of sufficient length
- 2 Complete excision of scar-tissue and removal of sclerosed bone until healthy vascular bone is exposed
- 3 Extensive surface of contact between graft and host-bone
- 4 The preparation of a healthy muscle bed
- 5 The avoidance of metallic or non-absorbable sutures for internal fixation of graft
- 6 Scrupulous attention to asepsis and perfect hæmostasis
- 7 Immobilization by plaster-of-Paris until firm osseous union has occurred between graft and host-bone



FIG 194
Tibia from Cas. 17
Before graft operation



FIG 195.—The same case as Fig 194. Graft survived severe latent infection. Callus thrown out from the ends of host and new bone creeping along the graft. Five months after graft operation.

In every case of ununited fracture the success or failure of the operative treatment will depend upon the correct appreciation of these factors. The inlay cortical graft as popularized by Albee has not proved so successful as may have been expected in ununited war fractures. The technique of this operation will not permit of the placing of a very broad piece in the fragments, and it was on account of many failures observed that the author was led to try a different type of operation. Practically all failures can be definitely attributed to technical errors, such as too small a graft, infection, or inadequate fixed bony approximation of the graft to the host-bone.

Before describing in detail the operation that has given excellent results, it is desirable to consider briefly certain important factors in regard to the bone-graft itself.

In my experience the autogenous tibial graft, including periosteum, compact bone, and medullary tissue, has proved most satisfactory. The bone is easy of access and from it a graft can easily be cut of any shape, length, or thickness required. Rapid regeneration of bone soon fills the gap left in the tibia, so that no permanent disability results. Occasionally a hematoma may develop, but no serious complications have ever occurred. As a rule the wound is strongly healed at the end of a fortnight.

In very few cases were grafts free of periosteum employed. The results of those in which the periosteum was not included were just as satisfactory. Although no reliance can be placed on the periosteum for production of bone, I am satisfied that it facilitates the secondary vascularization of the graft and also protects the graft in the event of the lighting up of latent sepsis. Stripping up of the periosteum during the course of an

operation should therefore be carried out with the greatest care, and to the minimum amount, to avoid underlying necrosis should infection ensue. It has been my practice, when intramedullary grafts were considered necessary, and employed successfully in 7 out of 10 cases, to remove the periosteum from that part of the graft which is fitted into the medullary cavity of the host-bone. Whilst the compact bone does not seem to take an active part in osteogenesis after transplantation, it supplies the strength to withstand the strain of function when union is complete.

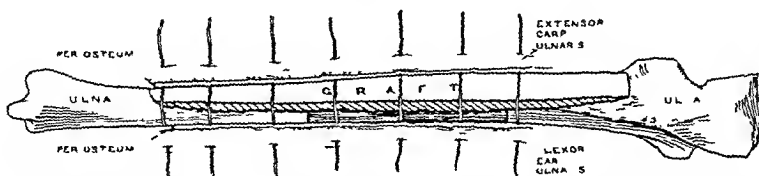


FIG 196 — Diagram showing autogenous massive tibial graft as employed in ununited fracture of ulna

The medullary tissue however, would appear to be the main route along which new bone formation extends between the fragments of the host-bone. It is, therefore, advisable to include as much as possible of this tissue in any graft (Fig 196).

The size of the graft is important. It must be cut long enough, not only to bridge the gap, but to have contact with a wide surface of the host bone on either side. The

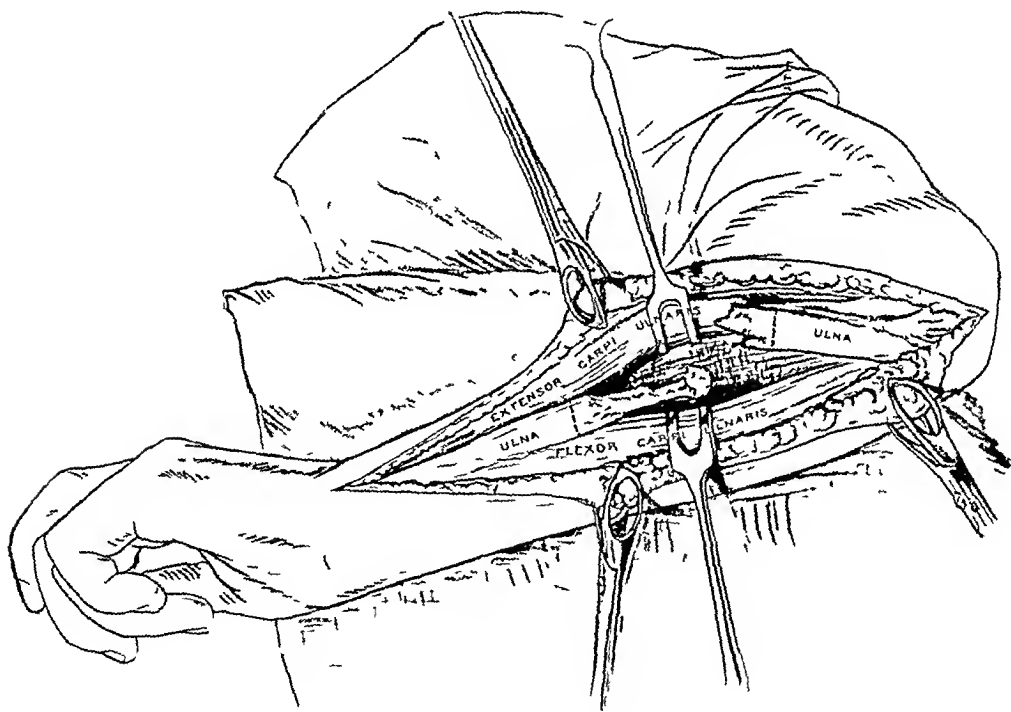


FIG 197 — Diagram showing skin incision and dissection to prepare ununited fragments of ulna and make a bed for reception of graft

usual length is found to be from two to three times that of the gap. This is an important technical point, and cannot be too strongly emphasized. The graft is seldom less than four inches. A long graft affords, not only better and firmer fixation, but also a larger surface of contact between the host and the graft, which increases the means of access for

the new blood-supply Further, the more the graft approximates in size to the bone to be replaced, the less liable it is to fracture, and the more quickly will full strength be obtained in the reconstructed limb

I now come to the operation which has engaged my special attention for some time past No claim is made as to its being entirely original in conception, but its application in a large number of ununited war fractures having proved so highly successful seems to justify my personal experience of its effectiveness being put on record

In my experience the autogenous graft obtained from the subcutaneous inner surface of the tibia, and employed as a massive lateral graft, has formed the most satisfactory method of dealing with non-union of the ulna, radius, and tibia following upon gunshot injuries The operation will be described as it is carried out for the ulna

The bone should be approached along its postero-internal border between the flexor and extensor carpi ulnaris Too great emphasis cannot be placed upon the importance of making a skin incision of sufficient length (*Fig 197*)

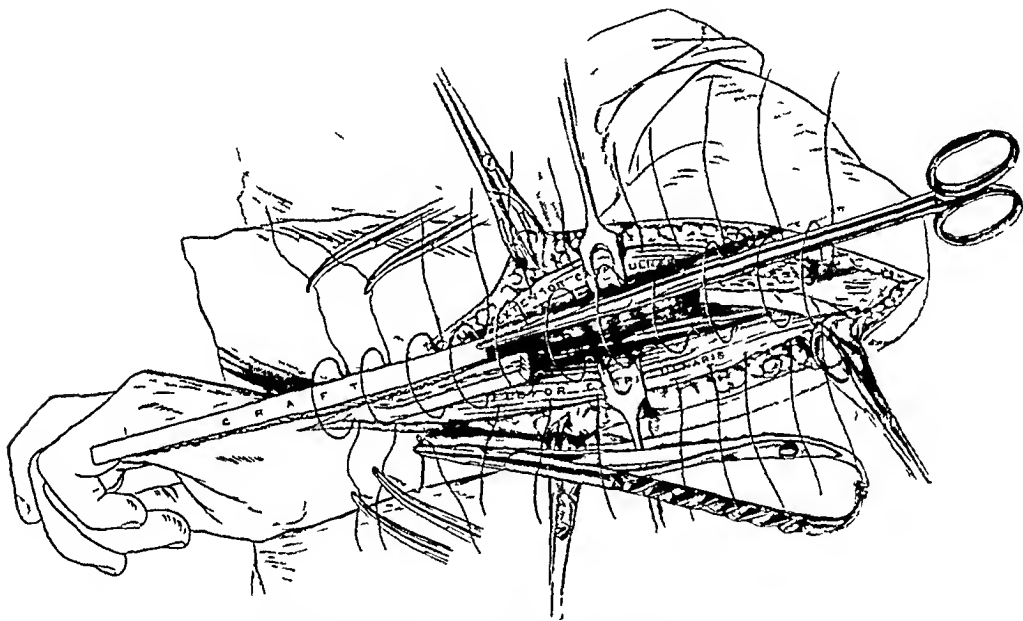


FIG 198.—Diagram of intraosseous graft and bed on postero-internal surface of ulna completed Method of placing graft under looped sutures of strong tanned catgut

The first step of the operation should be to excise all scar-tissue, whether in the skin or deeper tissues In regard to the former, this should be done as a preliminary operation whenever the skin cicatrix is extensively adherent to the underlying structures Otherwise, if a large skin scar has been left to cover the tissues, it will, within a few days, necrose in part and leave a troublesome superficial ulcer to heal The deep scar-tissue between the bone fragments must also be regarded as tissue of poor vitality, deficient circulation, and weak resistance Such a preliminary operation was carried out in 10 cases A bone-graft implanted in scar-tissue would most probably be absorbed, or at least atrophy, and fracture easily

In the next step of the operation the ends of the host fragments are exposed, and all sclerosed and ragged bone between the fragments removed until healthy vascular bone appears All sclerosed bone is of very low osteogenetic power The muscles, along with the periosteum, are then stripped from the bone for fully two inches from the fractured ends and for practically one-fourth of the circumference of the bone (*see Fig 197*) Next beginning in a direction away from the point of fracture, and extending the whole

circular saw (preferably single) electrically driven. While the saw is cutting it is constantly sprayed with saline solution. The transplantation should be made immediately. I am convinced that it is a mistake to wash the graft in saline lotion or leave it in saline while something else is being done. The best results follow immediate closure of the deep tissues round it, and suture of the surface wound. The graft is placed underneath the catgut loops as shown in Fig 198, and held in close apposition to the raw surfaces of the parent bone whilst the ligatures are being tied. A few additional catgut sutures are necessary to unite the surrounding muscles and so ensure a complete covering for the graft (Fig 200). The skin wound is closed with interrupted silkworm-gut stitches.

The limb must now be securely controlled in correct position by a plaster-of-Paris case, which is the only adequate post-operative dressing. It should be applied with the utmost care over a thin padding of cotton-wool or flannelette bandage, which fixes the wound dressing, moulded to the bony contours of the extremity, and should always include at least one joint above and one joint below the bone involved. In the forearm cases the position of the limb is important. The elbow is flexed to a right angle, and the forearm supinated as completely as possible. The limb should be held in the desired position throughout.

POST-OPERATIVE TREATMENT

The protection of the graft from undue stress subsequent to the operation is best attained, in the writer's view, by the application of a plaster-of-Paris casing from the fingers to the mid humerus and applied at the time of operation. The padding employed usually prevents any excessive swelling of the limb. However, should œdema develop, the cast ought immediately to be split down the whole length of the aspect furthest from the graft. The condition is quickly relieved and no harm is done. A plaster case may be made considerably lighter by reinforcing it at the points of special strain by wire or narrow strips of metal.

Absolute immobilization of the part involved is maintained for six weeks. During this period the graft is establishing a vascular continuity with the host at either end and with the surrounding tissues and it is not necessary to interfere with the plaster case.

After the expiration of six weeks the skin stitches are removed, and before a second plaster casing is applied the degree of union between the graft and host-bone is determined by a radiographic examination (Fig 201). Success is unlikely if the graft is not firmly united with the host at both ends.

In the case of the forearm the elbow is again flexed to a right angle, and a small window is cut on the anterior and posterior aspects of the forearm so that gentle faradic stimulation of the flexor and extensor muscles may be carried out. A small short cock up splint should be incorporated with the plaster, to permit the hand being left free for exercises and massage without straining the graft. Such measures unquestionably stimulate bone growth by allowing the graft to functionate as early as possible and within the limits of safety.

At the end of three months from the date of operation the plaster is dispensed with, and the nutrition of the limb is gradually restored by massage, faradism and active use.

The time involved by the change from the stage of partial function to that of complete function depends upon (1) The presence or absence of other serious disabilities, e.g., nerve lesion, muscle destruction etc.; (2) The state of union between the host-bone and graft; (3) The individual bone involved. The growth and union of the graft are easily estimated by radiograms which should be taken every six or eight weeks (Fig 201). During the transitional period it is advisable that the forearm be supported by the wearing of a short



FIG. 201.—(Line from Case 13.) Massive lateral tibia graft united strongly with host bone at both ends seven weeks after graft operation.

cock-up splint, and particular attention be given to exercises to encourage the return of the movements of pronation and supination. As regards the tibia, the plaster casing should be worn for a period of six months at least subsequent to operation, and for a further three months an external metal support is essential. Such conservatism will certainly avoid the occurrence of some failures.

CONSIDERATION OF INDIVIDUAL BONES

Radius—Non-union of the radius is more important than that of the ulna, owing to the considerable weakness of grasp resulting. It may occur in any part of the shaft, but is especially frequent in the lower half (*Fig 202*). In most cases a bone grafting operation is necessary. The hand is attached to and articulates mainly with the radius, so that loss of the support of the latter owing to non-union is associated with considerable weakness of grasp and with radial deviation of the hand, which deformity is kept up by contracture of the radial tendons, these stretching like a bow string across the gap in the bone (*Fig 203*). The lower fragment of the bone tilts towards the ulna and the hand is deviated towards the radial side, the styloid process lying at a higher level than that of the ulna (*Fig 204*). Where the lower fragment of the radius is less than an inch in length,



FIG 202—Radius from Case 39. Non-union from the loss of bone between the fractured ends. Marked deviation of distal fragment with consequent radial deviation of hand (see *Fig 203*). For result of grafting operation see *Figs 205* and *208*.



FIG 203—Case 39. Illustrates radial deviation of hand. Contracture of radial extensors of wrist well seen. For result after operation see *Fig 205*.



FIG 204—Case 19. Large portion of shaft missing and considerable deviation of distal fragment. For result of grafting operation see *Fig 211*.

shortening of the ulna to correct radial deviation and allow direct union of the radial fragments has been recommended. This procedure is not advisable as it is sometimes followed by non-union of the ulna. The hand deviation can be as satisfactorily corrected by lengthening of the contracted radial tendons (*Fig 205*).

There are often associated injuries of the tendons and muscles, particularly of the extensor muscles of the thumb. An injury to the median nerve is not an uncommon complication. Large adherent scars are also frequent, and are important because these may interfere with the success of an operation unless they can be completely removed at a preliminary operation.

The radius is best exposed along the line which separates the radial extensors of the



FIG 205



FIG 206



FIG 207

FIG 205—Case 29 Radial deviation of hand almost completely corrected by lengthening of contracted radial extensors of the wrist at grafting operation For result of bone graft see Fig 208

FIG 206—Case 34 Posterior subluxation of lower end of ulna, occasionally a complication of non union of radius in its lower third

FIG 207—Radius from Case 27 Modified intramedullary graft—pegging one end of graft into medulla and the other fitted into gutter Result 2 months after operation Ultimately strong union

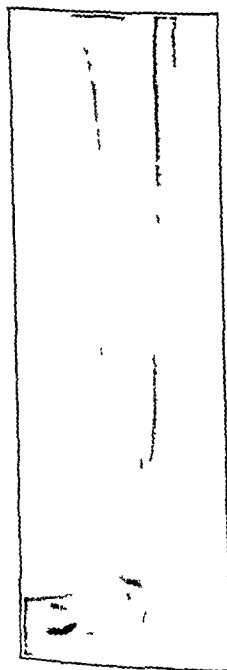


FIG 208

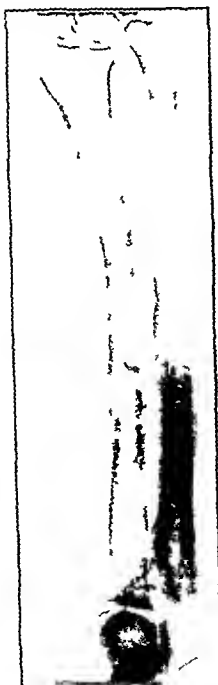


FIG 209



FIG 210



FIG 211

FIG 208—Case 29 Same as Figs 202 205 206 Final result 29 months after grafting operation for non union of radius August 1919 Medullary canal completely reformed Massive tibial graft employed

FIG 209—Case 30 Final result of tibial graft for non union of radius—three years after operation February 1919 Complete canalization of graft

FIG 210—Case 41 Final result of tibial graft for non union of radius—two years after operation January 1920 Canalization of graft almost complete

FIG 211—Case 15 Same as Fig 204 Final result of tibial graft for non union of radius—22 months after operation November 1919 Lengthening of flexor carpi radialis and brachioradialis in addition to radial extensors of wrist in plan I to correct extreme degree of radial deviation of hand

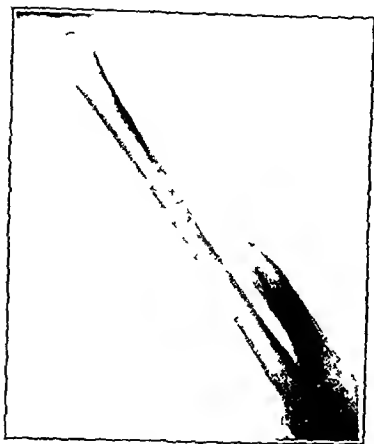


FIG 212—Ulna from Case 2. Large portion of shaft missing. For result of grafting operation see Fig 213

wrist from the *extensor communis digitorum*. In its distal third the *extensor pollicis brevis* and the *abductor pollicis longus* crossing the tendons of the radial extensors of the wrist often render access to the tilted lower fragment somewhat difficult, whereas exposure in the upper two-thirds is a comparatively simple dissection. To secure proper alignment it is necessary to lever the lower fragment away from the ulna and to rotate it into the supinated position. A slight radial deviation of the hand may remain, but this is not of importance. A posterior subluxation of the lower end of the ulna (Fig 206) is by no means a rare complication, and occasionally gives rise to pain in the neighbourhood when the patient has resumed his employment, and particularly if this is of a laborious nature.

The graft is preferably applied to the posterior surface of the host-bone. In fractures above the level of the insertion of the pronator radii teres, it is important to remember that whereas the proximal fragment is found completely supinated, the distal is fully pronated. In some cases when the site of non-union is close to the wrist-joint it is impossible to obtain a satisfactory bed and coverings for a lateral graft



FIG 213—Ulna from Case 3. Non union middle of shaft. For result of grafting operation see Fig 221



FIG 213a—Ulna from Case 3. Non union in upper third. Proximal fragment flexed and tilted towards the radius. For result of grafting operation see Fig 217



FIG 214—Ulna from Case 9. Condition of non union when patient admitted to Pringour Hospital

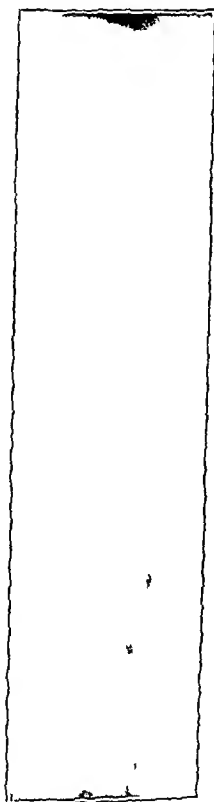


FIG 214a—Case 9. Same as Fig 214. In addition to missing tibial graft an intramedullary pc was employed to correct deviation of proximal fragment. For operation result see Fig 217



Fig. 217—Case 9. Same as Figs. 215, 216. Final result 15 months after double graft-intramedullary and lateral incision.



Fig. 218—Case 13. Same as Fig. 201. Final result of tibial graft for non-union in upper third of ulna—8 months after operation, September, 1921.

Firm fixation of the graft is also essential. For such cases a modified intramedullary peg proved successful. This method consisted of pegging one end of the graft into the medulla of the proximal fragment and fitting the other into a gutter made in the short distal fragment (Fig. 207).

Ulna—Non-union of the ulna is of frequent occurrence, but is much less important (Figs. 212–214). Whilst most ununited fractures of the radius require bone-grafting, some ununited fractures of the lower third of the ulna, or of the olecranon, cause so little disability that this is not necessary. When it occurs low down in the shaft it affects the utility of the hand comparatively little, and any deformity is slight.

There is usually no considerable displacement of the fragments to be corrected except when the

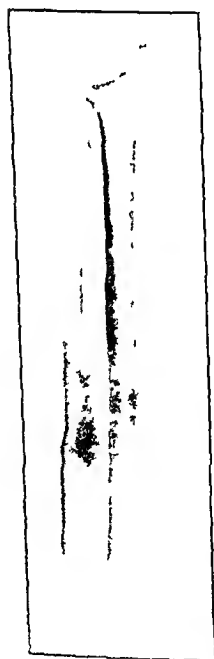


Fig. 219—Case 10. Same as Fig. 217. Final result 15 months after graft-intramedullary and lateral incision.



Fig. 220—Case 11. Same as Fig. 218. Result 1 month after operation.



Fig. 221—Case 12. Same as Fig. 219. Final result 15 months after graft-intramedullary and lateral incision for non-union of ulna.

fracture involves the upper third, the proximal fragment often being flexed and tilted towards the radius (*Fig 215*) It is advisable to correct this deviation and maintain proper alignment by an intramedullary peg (*Figs 216, 217*) this being additional to the usual lateral graft employed Preliminary excision of the skin cicatrix has been more frequently required in the case of the ulna

The ulna, being a less vascular bone than the radius, with usually a considerable thickness of sclerosed bone at the common site of non-union in the upper third, it is essential to prepare the best available musculo-osseous bed

It is rare to find both bones of the forearm ununited Shortening of both to allow direct union and a bone-graft of the ulna to ensure adequate fixation, is probably the best operative procedure

Humerus—The cases of non-union of the humerus numbered 19, and generally constituted a difficult surgical problem



FIG 222—Humerus from Case 81 Non union in middle third For operation result see *Fig 223*

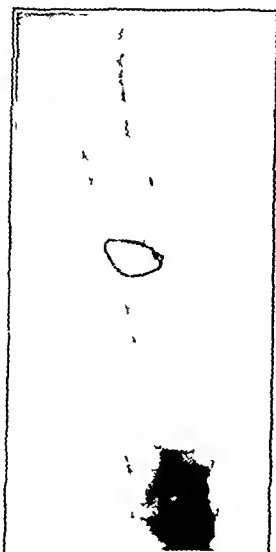


FIG 223 — Case 81 Same as *Fig 222* Result 8 months after step cut operation Good union



FIG 224—Humerus from Case 68 Non union in region of neck For result of operation see *Fig 225*



FIG 225—Case 68 Same as *Fig 224* Result two years after squaring of fragments intramedullary peg and sewing of iliac bone chip Good union Function of shoulder excellent



FIG 226—Humerus from Case 67 Non union in region of neck Result 22 months after intramedullary peg and squaring of fragments Additional fixation by wire in case of absorption of intramedullary graft Strong union Function of shoulder excellent

It has been stated that in proportion to the number of fractures sustained by this bone, non-union is more frequent than in any other bone in the body The chief reason for this relatively high occurrence is that fixation is particularly difficult to maintain Of

the patients, 2 had musculospiral paralysis at the time of operation, and 10 cases had been previously operated on elsewhere, not infrequently as often as three times

Freshening the ends of the bone and fixation by wiring, plating, and inlay grafting had been practised, and with persistence of



FIG 227—Humerus from Case 87 Non union in region of neck treated by removal of sclerosed bone impaction of distal fragment into the proximal and obturator, internal fixation by kangaroo tendon Result 7 months after operation Strong union Function of shoulder good



FIG 228—Humerus from Case 94 Double graft employed Absorption of intramedullary graft For final result see Fig 229

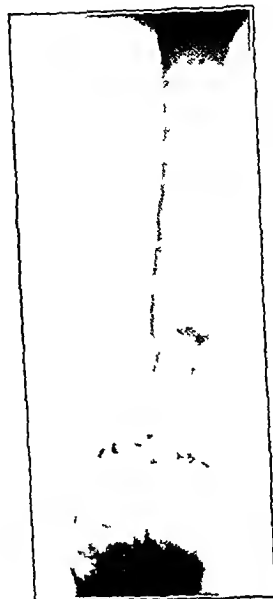


FIG 229—Case 94 Same as Fig 228 Result 16 months after grafting operation Strong union

non-union, which is found to occur frequently in the lower third of the shaft These results clearly indicate the unsatisfactory nature of the operative procedure employed

in the early humerus cases Whilst all are agreed that the autogenous bone-graft has given the best results in the radius, ulna, and tibia, it cannot be employed so generally in the case of the humerus Bone-grafting is of little value in filling gaps in the shaft of the humerus Ununited fracture of the humerus is most certainly remedied by the step cut operation, and the steps should be long (Figs 222 and 223) Shortening of the arm is of minor importance This method was successfully employed in 9 out of 10 cases

Alternative operative measures are best determined according to, (1) The site of non-union, (2) The gap in the bone (3) The condition of the neighbouring joints Non-union in the region of the neck can be successfully treated (Fig 224) (1) By freshening the ends of the fragments employing an intramedullary



FIG 230—Humerus from Case 97 Non union in lower third For result of rifting see Fig 231



FIG 231—Case 97 Same as Fig 230 Final result 9 months after grafting Very strong union Function of elbow excellent

peg as an internal splint, and sowing in the fracture site small chips of bone obtained from the iliac crest (Figs 225, 226), or (2) After thorough removal of sclerosed bone,

impacting the distal fragment into the proximal portion and obtaining fixation by wire or kangaroo tendon (Fig 227)

Ununited fractures of the lower third of the humerus complicated by ankylosis of the elbow-joint are undoubtedly amongst the most difficult cases to treat. The reason for this lies in the real difficulty of providing adequate internal fixation of the fragments. This is more especially the case when the distal fragment is short, tapering, and brittle. The step-cut operation is generally not advisable, but combined with Pirham's metal bands the desired result can be obtained. Equally satisfactory results have followed the use of the intramedullary peg supplemented by a lateral graft or chips of iliac-crest bone (Figs 228, 229, 230, 231)

Whichever operative measures are adopted, it is essential to fix the whole arm and chest in plaster-of-Paris at the time of operation. The arm is abducted to about a right angle, and the elbow is flexed, this being done to prevent any undue strain upon the fracture site. Fixation in this position is maintained for about eight weeks, at the end of which period the stitches are removed and the plaster cast is replaced by a suitable splint should the radiograms show that strong osseous union has occurred. In the majority of cases, however, it is safer to employ a plaster cast for a period of twelve to sixteen weeks. This need not interfere with the postoperative treatment, as the arm portion of the plaster-cast can be bivalved.

Tibia—The following groups may be distinguished—(1) *Fractures of the tibia with fibula intact*, (2) *Fractures of the tibia with old fracture of fibula at opposite point*

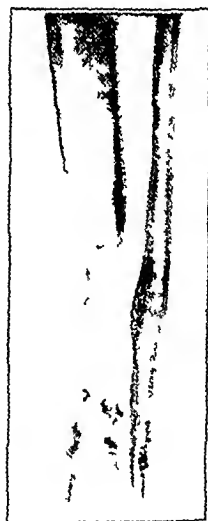


FIG. 232.—Tibia from Case 61. Non-union with 2 in. gap. Transverse fracture of fibula at opposite point. For result of graft see Fig. 230.



FIG. 233.—Tibia from Case 77 before operation. Non-union with lateral displacement of upper fragment. For result of grafting see Fig. 234.

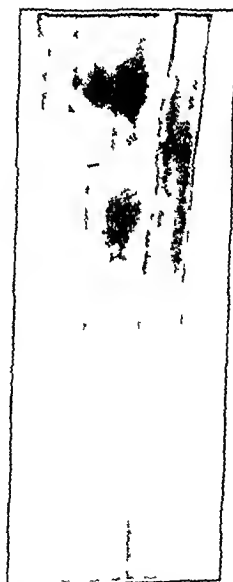


FIG. 234.—Case 73. Same as Fig. 233. Result 1 month after introduction of two major grafts from opposite tibia. Grafts survived violent latent infection. Strong union.

Group 1—There is usually very little displacement and the rigid intact fibula prevents apposition of the fracture ends. The line of fracture is transverse or slightly oblique. The primary loss of osseous tissue is small so that a single lateral graft suffices. The operative technique is similar to that carried out in the case of the ulna.

Group 2—Whilst fracture of the tibia is of the large short splinter type followed by chronic osteomyelitis and necrosis with a resultant gap of $\frac{1}{2}$ to 2 inches, that of the fibula

has been transverse (Figs 232, 235). The displacement is always more marked in the tibia when the fibula has been simultaneously involved. There is frequently an angular displacement, the leg appearing to form a curve with a marked anterior convexity. In addition, some lateral displacement is generally present (Fig 233). The lower fragment usually shows a certain degree of rotation on the long axis of the bone, resulting in internal or external displacement of the foot and almost always a degree of pes equinus. In consequence of extensive sear-ing of overlying skin and also muscular injury, bone-grafting may be a very difficult procedure. It is beneficial in all such cases to carry out at a preliminary operation a complete excision of all superficial and deep sear-tissue at the site of non-union. About a month later the grafting operation is performed. It is advisable to insert two massive lateral grafts whenever possible (Fig 234) in order to induce more rapid and entire replacement of the bony loss and to increase the strength of the resulting union. Unfortunately, re-fracture is by no means a rare occurrence and for this reason there should be no undue haste in dispensing with the plaster casing, which should be worn for a period of six months at least subsequent to operation. For three months more the patient wears an external metal support.



FIG. 232—Case of same as Fig. 231. Six months after grafting operation. Strong union. Function of leg good.

Femur—Whilst delayed union of the femur is frequent, non-union is of very rare occurrence. In fact, the diagnosis of non-union should not be made unless, after a prolonged trial (not less than twelve months) of conservative measures (Thomas splint, 'limb-casting' and damming, ionization, fridism, and diathermy), there is no evidence of union either clinically or in radiograms.

The site of delayed union and non-union in all three cases of the present series was about the middle third of the shaft. When non-union does occur in this region it appears to be due to latent sepsis. Invariably the infection has spread to the knee-joint, whose movements become so limited that there is almost a fibrous ankylosis. In short, the function of the limb is considerably reduced.

The operation for non-union of the femur is at all times a serious one. Only one of the femur cases under review required operative interference, this consisting of freshening the fracture ends, complete excision of sclerosed bone and of intervening fibrous tissue, insertion of an intramedullary peg, and additional fixation by wire. The whole limb, including the pelvis, is put up in plaster-of-Paris for three weeks or a month after which period a Thomas splint with extension is substituted.



FIG. 236—Radius from Case 28. Early fracture of intramedullary peg. Strong union of graft with proximal fragment but non-union with distal fragment. Strict and prolonged immobilization failed to bring about union.

Graft Fractures—In the series of 83 cases subjected to the operation of transplantation of bone, 7 graft fractures occurred—3 in the ulna, 2 in the tibia, and 1 each in the radius and humerus. All but one were massive tibial grafts, and the fractures occurred at a late stage in 6 of the cases after osseous union between the host-bone and graft was complete. The exceptional case was an intramedullary peg, which at an early stage showed firm union of host-bone, but a false joint at the distal end (Fig 236). The opposite the junction of the graft and the host-bone. Such a fracture may even occur inside a well-fitting plaster case, and is due to inadequate contact between the graft surface and the medullary tissue of the host-bone. If an intramedullary peg has been employed, the site of fracture becomes a typical non-union, the extremity of the fracture

becoming peg-shaped (*Fig 236*) After the fracture is complete, little further absorption appears to take place Strict immobilization after the fracture was discovered failed to bring about union in the case shown in *Fig 236*

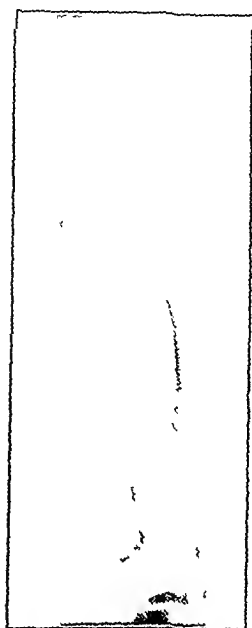


FIG 237

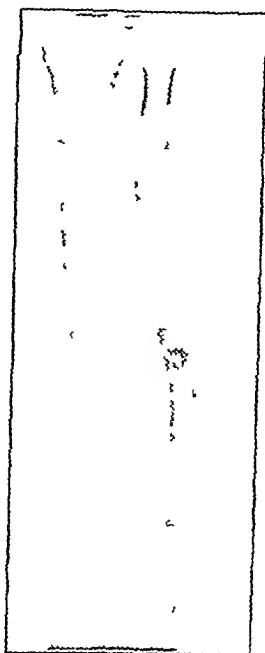


FIG 238



FIG 239

Late fractures occur several months after the bone grafting operation. Their site is almost always about the middle of the graft. The graft has been entirely successful, being firmly united at both ends, and the host-bone almost entirely replaced. The patient has probably returned to his civil occupation and as a result of some strain, the fracture is produced (*Figs 237-241*). Many cases will reunite, although a prolonged period is required for the union to take place.



FIG 240

FIG 237—Ulna from Case 12. Traumatic or late graft fracture through middle of graft six months after successful graft operation.

FIG 238—Case 12. Same as Fig 237. Result 8 months after accident—graft fracture strongly united. Well marked callus thrown out.

FIG 239—Ulna from Case 4. Traumatic or late fracture through middle of graft six months after successful graft operation.

FIG 240—Case 1. Same as Fig 239. Result 10 months after accident—graft united but no resultant disability.

FIG 241—Tibia from Case 62. Late graft fracture about middle of graft occurred seventeen months after successful graft operation. Strong union resulted from second graft operation.

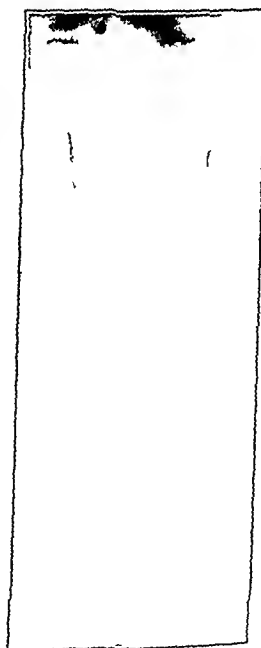


FIG 241

Table II—ANALYSIS OF RESULTS

BONE	NUMBER OF CASES	FAILURES	PARTIAL UNIONS	COMPLETE UNIONS	CASES NOT REQUIRING OPERATIVE INTERFERENCE
Ulna	36	0	0	30	6
Radius	26	1	1	20	4
Humerus	19	1	2	16	0
Tibia	15	1	0	10	4
Femur	3	0	0	1	2
Fibula	1	0	0	0	1
Total	100	3	3	77	17

SUMMARY AND CONCLUSIONS

The series of cases studied was unselected and comprised 100 patients with ununited fractures due to war injuries. Of the 100, 83 required operative treatment. Of these, 70 were subjected to the operation of autogenous bone-grafting. In 67 (95.8 per cent) the grafting operations were successful, and 3 (4.2 per cent) were failures. The cause of failure was latent infection, and the graft was lost in consequence—in 2 of the cases with persistence of non-union. In the series of 83 operations there were 6 cases of latent infection. These six cases had been operated on previously, and all but one had had infection previously. The cause of infection depends upon the type of case rather than on the operative technique. It is probable that the general adoption of a two stage operation would lower the percentage of infections. However, infection of a wound and a successful graft are not incompatible. In 61 cases the operation carried out has been a primary one, and in 24 cases unsuccessful attempts had been previously made by other surgeons.

Autogenous massive grafts obtained from the subcutaneous inner surface of the tibia have proved very successful, particularly in the ulna, radius, and tibia. They should be of good size—as large as the bone which is being replaced, and two to three times as long as the gap. The more the graft approximates in size to the bone it is to replace, the less liable it is to fracture. The medullary tissues would appear to be the main route along which new bone formation extends between the fragments of the host-bone. It is therefore advisable to include as much as possible of this tissue in any graft. Compact bone is required for strength to withstand the strain of function when union is complete. The presence or absence of periosteum on the graft does not appear to affect the vitality or growth of the graft. Internal fixation of the graft is most essential for a successful result, and depends to a considerable extent upon accurate suturing. Interrupted and 'looped' sutures of strong tanned catgut have been employed throughout the series almost without exception. The only adequate post operative dressing is a plaster-of-Paris case applied at the time of operation. Absolute immobilization of the part involved is maintained for six weeks, during which period firm union should occur. The degree of union between the graft and host-bone can be determined by frequent radiographic examinations. During the transitional period adequate support (plaster or splint) of the graft is essential.

The bone-graft as usually employed in the intramedullary and inlay methods is too small, and not suited for ununited fractures due to war injuries.

In conclusion, my thanks are due to Mr A. F. McConnochie, and also Dr John W. L. Spence and Mr J. McGill of the Radiological Department of the Ministry of Pensions Hospital, Crugleith, for the radiographic prints. The drawings illustrating the operative treatment are the work of Mr J. T. Murray, to whom I am indebted for the great care expended in their preparation.

SYNOPSIS OF 100 CASES OF UNUNITED FRACTURES DUE TO

CASE	DATE OF WOUND AND WHEN HEALED	CAUSE AND PERIOD OF NON UNION	BONE AND SITE OF NON UNION	PREVIOUS OPERATION
Case 1—C A	Sept 1917 July, 1919	1 in gap 25 months	L ulna Middle third	Sequestrectomy and excision of abscess
Case 2—A B Figs 212, 219	Oct 1917 April, 1918	3½ in gap 17 months	R ulna Lower half Fragment of ulna remaining, 1½ in	Fusion of scar and free tendons
Case 3—R B Figs 213, 220	Sept 1918 Jan 1919	1 in gap 10 months	R ulna Middle third	Suture of median nerve
Case 4—J B Figs 239, 240	April 1918 Sept 1918	2 in gap 33 months	L ulna Middle third	Suture of median nerve
Case 5—G C Figs 215, 216, 217	April, 1918 Aug 1918	2 in gap 30 months	L ulna Upper fourth	Suture of ulnar nerve
Case 6—J D	Oct 1918 Feb 1919	1 in gap 16 months	L ulna Middle third	Nil
Case 7—S McL	June, 1918 Aug 1918	3 in gap 27 months	L ulna Middle third	Removal of F B
Case 8—A M	May, 1917 Sept 1917	1 in gap 25 months	R ulna Middle third	Freeing of median nerve
Case 9—N S Figs 214, 221	April, 1915 Nov 1917	1½ in gap Sclerosis and plating 44 months	L ulna Middle third	23 operations for removal of dead bone, also flat ulna which was subsequently removed
Case 10—G T	June, 1918 Sept 1919	2½ in gap 18 months	L ulna Middle third	Nil
Case 11—T S	Nov 1918 Dec 1918	1½ in gap 13 months	L ulna Upper fourth	Three preliminary sequestrectomies
Case 12—J J Figs 237, 238	Sept 1915 Aug, 1916	1 in gap Sclerosis and wiring 64 months	L ulna Middle third	Sliding graft and fixation wire. Wire subsequently removed
Case 13—J A Figs 201, 218	Sept 1918 Nov 1918	1½ in gap 36 months	R ulna Upper fourth	Sequestrectomy
Case 14—T M	May 1918 Aug 1920	4 in gap 41 months	L ulna Middle third	Preliminary excision of
Case 15—A T	Sept 1918 April, 1919	2½ in gap 37 months	L ulna Middle fourth	Nil
Case 16—T W	Oct 1917 Mar 1918	2 in gap 47 months	L ulna Middle third	Nil
Case 17—H B Figs 194, 195	Sept 1918 Jan 1920	1½ in gap 41 months	L ulna Middle third	Sequestrectomy. No June 1920 which had removed on account of
Case 18—A C	Aug 1918 Jan 1919	Sclerosis and wiring 37 months	I radius Junction lower third and upper two thirds	Three operations (2 w/ ununited fracture

INJURIES, WITH END RESULTS OF OPERATIVE TREATMENT

DATE AND TYPE OF OPERATION	RESULT	REMARKS
Oct 1919 Tibial graft	Success	Operation delayed owing to persistent sinus and slow formation of sequestra. Complete range of pronation and supination. Resumed pre war occupation.
Mar 1919 Tibial graft	Success	Exceptionally long graft required—6 inches. Fracture of graft detected Feb 1920—strongly united Mar 1921. Marked formation of callus. When the fracture was detected patient was not aware of anything wrong with forearm. Range of movement between $\frac{1}{2}$ complete supination and $\frac{1}{4}$ complete pronation. Resumed employment as a joiner.
July, 1919 Tibial graft	Success	Range of movement between complete supination and the mid position. Employed as a rabbit trapper.
Jan 1921 Tibial graft	Success	Six months later fell and injured his arm. Fracture was exactly in middle of graft, no callus thrown out, no movement elicited at site of graft fracture. As patient felt arm just as strong as before the fracture no further operation was performed. Range of movement between $\frac{1}{2}$ complete supination and 10° from the mid position. Employed in a garage.
Oct 1920 able tibial graft external and intra medullary	Success	Forearm range of movement between $\frac{1}{2}$ complete supination and the mid position. Employed as a caretaker, and contemplates farming in Canada, for which he is quite fit.
Feb 1920 Tibial graft	Success	Some superficial erosion of graft in early x-rays. Forearm range of movement between complete supination and $\frac{1}{2}$ complete pronation. Resumed pre war occupation as a mason.
Sept 1920 Tibial graft	Success	Forearm range of movement between complete supination and 10° of pronation from the mid position. Employed as a postman.
June 1919 Tibial graft	Success	Forearm range of movement between complete supination and the mid position. Employed as a clerk.
Dec 1919 Tibial graft	Success	Forearm range of movement between complete supination and $\frac{1}{4}$ complete pronation. Unemployed. Does not think he will be fit for pre war occupation as a miner.
Dec 1919 Tibial graft	Success	Forearm range of movement between complete supination and 10° pronation from the mid position. Employed as an engineman.
Dec 1919 Tibial graft	Success	Forearm range of movement between $\frac{1}{4}$ complete supination and the mid position. Employed as a postman and finds his arm as strong as previously.
Jan 1921 Tibial graft	Success	Injured reconstructed forearm six months after operation—fracture of graft, which was found to be strongly united three months later. Employed as a miner.
Sept 1921 Tibial graft	Success	Forearm range of movement between complete supination and 10° of the mid position. Discharged from hospital to commence out patient treatment.
Oct 1921 Tibial graft	Success	Forearm range of movement between complete supination and $\frac{1}{2}$ complete pronation.
Oct 1921 Tibial graft	Success	Also had fracture of radius at same level but strongly united with fair alignment. Forearm range of movement between $\frac{1}{2}$ complete pronation. Still receiving massage and electricity.
Sept 1921 Tibial graft	Success	Fitted to resume employment in brick works. Forearm range of movement between $\frac{1}{2}$ complete supination and the mid position. Still receiving massage and electricity.
Feb 1922 Tibial graft	Success	Severe latent infection followed second bone graft—both forearm and leg—no damage to graft or tibia resulted. Still receiving massage and electricity.
Sept 1921 Tibial graft	Success	Forearm range of movement between $\frac{1}{2}$ complete pronation and $\frac{1}{2}$ complete supination. Still unemployed.

Continued on next page

SYNOPSIS OF 100 CASES OF UNUNITED FRACTURES DUE TO

CASE	DATE OF WOUND AND WHEN HEALED	CAUSE AND PERIOD OF NON-UNION	BONE AND SITE OF NON-UNION	PREVIOUS OPERATION
Case 19—W B Figs 204, 211	July, 1917 Mar, 1918	3 in gap 28 months	L radius Middle third	Freeing of median nerve
Case 20—W D Fig 209	Oct 1917 Feb 1918	1½ in gap 16 months	L radius Middle third	Nil
Case 21—R M	Aug 1917 Feb 1918	Latent sepsis and sclerosis 31 months	R radius Lower third	Freeing of flexor mu. Suture of median nerve
Case 22—A M	Aug 1918 Mar 1919	1 in gap 15 months	L radius Junction upper and middle thirds	Removal of F B
Case 23—A M	Mar 1918 July, 1918	2½ in gap 12 months	L radius Junction upper and middle thirds	Sequestrectomy
Case 24—A M	Oct 1917 Dec 1917	¾ in gap 17 months	L radius Junction middle and lower thirds	Nil
Case 25—A P	June, 1918 Nov 1918	3½ in gap 13 months	L radius Middle third	Freeing extensor muscle forearm Transplant
Case 26—J S	Oct 1917 Dec 1917	½ in gap Sclerosis and wiring 12 months	L radius Junction upper and middle thirds	Wiring of fracture
Case 27—R S Fig 207	April, 1917 Dec 1917	½ in gap Sclerosis 36 months	R radius Junction lower fifth and upper four fifths	Excision of scar and com- of radial deviation of by plaster of Paris
Case 28—T C Fig 236	Sept 1917 May, 1918	1½ in gap 30 months	R radius Lower fifth	Nil
Case 29—J C	April 1917 Dec 1918	1½ in gap 31 months	P radius Through lower third	Nil
Case 30—H C	June 1915 Feb 1918	1½ in gap 45 months	L radius Lower third	Suture of median nerve lower third of forearm
Case 31—A M	April, 1917 Sept 1918	½ in gap 20 months	L radius Lower fourth	Nil
Case 32—R McK	Sept 1918 Jan 1919	1½ in gap 10 months	R radius Junction lower and middle thirds	Freeing of median nerve extensor muscles of forearm
Case 33—P G	April, 1915 Oct 1918	Sclerosis ½ in gap 55 months	L radius Middle third	Nil
Case 34—J K Fig 206	May, 1917 Aug 1917	Sclerosis No gap 29 months	R radius Junction lower and middle thirds	Nil
Case 35—D D	Aug 1918 April 1919	Sclerosis ½ in gap 40 months	L radius Junction lower and middle third	Plating of radius P subsequently removed union resulted

INJURIES, WITH END-RESULTS OF OPERATIVE TREATMENT—continued

DATE AND TYPE OF OPERATION	RESULT	REMARKS
Nov 1919 Tibial graft	Success	Extensive removal of sclero-ed bone necessitated graft 6 in long Forearm movements between mid position and $\frac{1}{2}$ complete pronation Employed as a clerk
Feb 1919 Tibial graft	Success	Forearm range of movement between $\frac{1}{2}$ complete supination and $\frac{1}{2}$ complete pronation Able to resume pre war employment as a printer
Mar 1920 Tibial graft	Success	Forearm range of movement from mid position to 15° pronation Employed as a telephone operator
Nov 1919 Tibial graft	Success	Forearm held almost in complete supination Low degrees of movement possible Almost complete canalization of graft
Mar 1919 Intra medullary graft from radius	Success	Forearm range of movement between complete supination to 10° from the mid position Employed as a clerk
Mar 1919 Sliding graft from radius	Success	Ulna was fractured at opposite point and strongly united without operation Forearm range of movement between $\frac{1}{2}$ complete supination and the mid position Resumed pre war occupation as a miner
July 1919 Tibial graft	Success	Septic dermatitis of arm so persistent that amputation was advised This condition however, was cured by lipoid paraffin Forearm range of movement between complete pronation and the mid position Resumed pre war occupation as railway clerk
Oct 1918 Tibial graft	Success	Forearm range of movement between complete supination and $\frac{1}{2}$ complete pronation Complaints of pain in the lower end of the ulna, this being due to subluxation which may have resulted from attempting heavy work as a labourer
April 1920 Intra medullary tibial graft	Success	This type of graft employed on account of inadequate coverings for a lateral graft Employed in an office
Mar 1920 Intra medullary tibial peg	Partial success	Upper end strongly united, lower end failed to unite This type of graft was employed on account of inadequate coverings for a lateral graft Training as a picture frame maker
Refused operation Nov 1919	—	—
Mar 1919 Shortening of ulna (1½ in removed) wiring of radius and ulna	Failure	Radial deviation of hand corrected, and consequently able to work as a labourer Almost a new wrist joint formed at site of ununited fractures Strong flexion and extension present
Dec 1918 Graft operation attempted but impossible owing to hemorrhage	—	Radial deviation of hand very marked Posterior subluxation of lower end of ulna Unable to resume work as miner, but found suitable employment
July 1919 Tibial graft	Success	Forearm range of movement between $\frac{1}{2}$ complete supination and 10° of pronation from the mid position Unable to resume pre war occupation in pits, but has secured suitable work
Operation inadvisable owing to mal union at the opposite point Nov 1919	—	Quite a serviceable arm, although not fit for pre war occupation as a tailor
United without operation whilst awaiting admission to hospital Sept 1919	—	Forearm range of movement between complete supination and $\frac{1}{2}$ complete pronation Also had posterior subluxation of the lower end of the ulna Employed as a labourer
Dec 1921 Tibial graft	Success	Preliminary operation for removal of plate and correction of radial deviation of hand Forearm range of movement between $\frac{2}{3}$ complete supination and $\frac{2}{3}$ complete pronation Still receiving massage and electricity

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SYNOPSIS OF 100 CASES OF UNUNITED FRACTURES DUE TO W

CASE	DATE OF WOUND AND WHEN HEALED	CAUSE AND PERIOD OF NON-UNION	POSIT. AND SITE OF NON-UNION	PREVIOUS OPERATION
Case 36—J M	Nov 1915 Feb 1919	1 in gap 72 months	L radius Junction lower and middle thirds	Bone graft in another leg which was subsequently moved owing to Excision of scar
Case 37—W H	April, 1917 Feb 1919	Sclerosis 55 months	L radius Junction lower fourth with remainder	Sequestrectomy. Pl. operation removal of as no union resulted
Case 38—C McG	Aug 1917 Oct 1917	1 1/2 in gap 23 months	I radius Middle third	Nil
Case 39—G G Figs 202, 203, 205, 208	Sept 1918 Dec 1918	1 1/2 in gap 11 months	R radius Lower third	Nil
Case 40—D G	May 1918 Nov 1918	2 1/2 in gap 20 months	L radius Middle third	Sequestrectomy
Case 41—J B Fig 210	Oct 1918 Dec 1918	1 1/2 in gap 15 months	L radius Junction lower and middle thirds	Nil
Case 42—J B	Nov 1916 Nov 1917	Sclerosis 1 in gap and plating 34 months	L radius Middle third	Plating operation. Rem. of plate. Freeing of nerve
Case 43—A N	July, 1916 Mar 1917	1 1/2 in gap Marked sclerosis 69 months	R ulna Middle third	Sequestrectomy
Case 44—J C	Aug 1917 Dec 1917	2 1/2 in gap Displacement upper fragment 56 months	L ulna Upper third	Suture of ulnar nerve. E. cision of scar
Case 45—J McI	April 1917 June, 1917	1 1/2 in gap 58 months	I ulna Junction upper and middle thirds	Excision of scar
Case 46—J C	Aug 1917 Nov 1917	1 1/2 in gap 14 months	R ulna Junction lower and middle thirds	Nil
Case 47—L M	April, 1917 Oct 1917	1 in gap Still ununited	L ulna Junction lower and middle thirds	Sequestrectomy. Free. extensor tendon in fl.
Case 48—P C	Feb 1918 Nov 1918	1/2 in gap Still ununited	L ulna Junction lower and middle thirds	Nil
Case 49—D S	Dec 1917 Sept 1918	3 in gap Synostosis between upper end of ulna and opposite point of radius	R ulna Upper 3 in with olecranon process missing	Nil
Case 50—A S	Oct 1915 Sept 1916	Sclerosis 1 in gap	I ulna Lower fifth	Nil
Case 51—F B	May 1918 Dec 1918	1 in gap Still ununited	L ulna Lower fourth	Suture of median n. Sequestrectomy of ulna

INJURIES, WITH END-RESULTS OF OPERATIVE TREATMENT—*continued*

DATE AND TYPE OF OPERATION	RESULT	REMARKS
Nov 1921 Tibial graft	Success	Forearm range of movement between $\frac{3}{4}$ complete supination and $\frac{1}{2}$ complete pronation Is receiving massage and electrical treatment
Nov 1921 Tibial graft	Success	Still in hospital Forearm range of movement between complete pronation and $\frac{1}{2}$ complete supination
July, 1919 Tibial graft	Success	Forearm range of movement between $\frac{3}{4}$ complete supination and 10° of pronation from the mid position Employed as a labourer, and when working feels pain in inferior radio ulnar joint
Aug 1919 Tibial graft Marked adial deviation of hand corrected by lengthening of muscles	Success	Forearm range of movement between complete supination and the mid position Employed as a glass blower
Jan 1920 Tibial graft	Success	Forearm held in position of $\frac{1}{2}$ complete supination Very good grip Resumed pre war occupation in paper mill
Jan 1920 Tibial graft	Success	Forearm range of movement between complete supination and $\frac{3}{4}$ complete pronation Employed as a traveller
Sept 1919 Tibial graft	Success	Forearm range of movement between complete supination and the mid position Employed as a motor driver
April 1922 Tibial graft	Success	Still under treatment
April, 1922 Double tibial graft	Success	Still under treatment for ulnar nerve paralysis Employed as a caretaker
Feb 1922 Tibial graft	Success	Still under treatment
Oct 1918 Tibial graft	Success	Employed as a clerk
No operation	—	As site of fracture immediately above lower end of ulna, grafting operation considered inadvisable Employed as a printer
No operation	—	Operation not advised owing to site of non union Presumed pre war employment as a miner, and can lift a 56 lb weight with injured arm
No operation	—	As range of all movements voluntary power of muscles, and stability of joint all good, operation not advised
No operation	—	Operation not advised as patient suffered little disability from fracture
No operation	—	Operation not advised as site of fracture low down

Continued on next page

SYNOPSIS OF 100 CASES OF UNUNITED FRACTURES DUE TO W

CASE	DATE OF WOUND AND WHEN HEALED	CAUSE AND PERIOD OF NON-UNION	BONE AND SITE OF NON-UNION	PREVIOUS OPERATION
Case 52—A M	Aug 1918 Mar 1919	1½ in gap 44 months	L ulna Junction upper fourth and lower three fourths	Bone graft of radius
Case 53—R T	Aug 1918 Jan 1920	2½ in gap 17 months	R ulna Middle third	Sequestrectomy
Case 54—D W	April 1917 April 1918	Sclerosis and latent sepsis 58 months	R femur Middle third	Sequestrectomies Several secesses
Case 55—G D	April 1917 Aug 1919	Sclerosis ¾ in gap	R tibia Junction lower and middle thirds	Nil
Case 56—J B	April, 1918 Mar 1919	2 in gap 11 months	R tibia Middle third	Sequestrectomies Fit bone graft, excision of in other hospitals
Case 57—W C	Oct 1918 June, 1919	Sclerosis and displacement 17 months	R tibia Middle third	Nil
Case 58—P S	Aug 1917 Sept 1918	¾ in gap 21 months	R tibia Middle third	Nil
Case 59—B W	May, 1915 Jan 1916	Sclerosis Still ununited	L tibia Middle third	Nil
Case 60—J McL	April, 1917 June 1920	Sclerosis 38 months	L tibia Middle third	Nil
Case 61—T H	Sept 1918 Mar 1921	2 in gap 30 months	I tibia Junction middle and upper thirds	Excision of skin scar and scar tissue
Case 62—H K A Fig 241	May, 1917 April, 1918	2 in gap 33 months	R tibia Junction upper and middle thirds	Excision of 1 in scar tissue
Case 63—J G	May, 1915 June 1919	1½ in gap 62 months	R tibia Through middle third	Excision of wound and tibia removal of plate
Case 64—I F Figs 232, 235	Oct 1917 June 1919	2½ in gap 22 months	I tibia Middle third	Sequestrectomy
Case 65—C R	Mar 1918 Nov 1918	Sclerosis and displacement 51 months	L humerus Junction of lower and middle thirds	Exploration of muscul nerve Transplantation tendons
Case 66—J McK	Mar 1918 Aug 1918	Sclerosis 24 months	I humerus Junction lower fifth and upper four fifths	Suture of muscul spiral Suture of ulnar nerve graft of humerus subsequently removed epio spiral nerve re
Case 67—H B Fig 226	Oct 1918 Mar 1919	Sclerosis 12 months	P humerus Anatomical neck	Unsuccessful bone other hospital
Case 68—A R Figs 224, 225	Oct 1918 June 1919	Sclerosis 16 months	R humerus Surgical neck	Sequestrectomy and of sinus

WOUNDS AND INJURIES, WITH END-RESULTS OF OPERATIVE TREATMENT—continued

DATE AND TYPE OF OPERATION	RESULT	REMARKS
April, 1922 Tibial graft	—	Still in hospital Almost complete canalization of radius graft
Jan 1920 Tibial graft	Success	Forearm range of movement between complete supination and $\frac{3}{4}$ complete pronation Resumed employment in general motor work
United without operation Feb 1922	—	Owing to the persistence of latent sepsis, the question of operation could never be considered Treatment consisted of (1) Extension by Thomas's splint (2) 'Hammering and damming' (3) Calcium ionization and calcium salts internally (3) Endocrine gland tablets, (4) Paradism
United without operation	—	Osteogenesis was delayed by sepsis
Mar 1919 Tibial graft	Failure	Graft fixed by wire partial death of graft this portion removed along with wire Non union again resulted
Mar 1920 Tibial graft	Success	Unemployed Still wears a steel support, as he is afraid to bear his whole weight on reconstructed limb
Mar 1919 sliding tibial graft	Success	Would have been able to resume pre war occupation as a miner but for 3 in shortening of the leg as a result of fracture of the femur Wears a high boot Employed as a motor man in the mines
No operation	—	Refused treatment
United without operation	Success	'Hammer and dam' treatment
Dec 1921 Double tibial graft	Success	Portion of host tibia was utilized when filling up gap between the two grafts Although still under treatment firm union has resulted
Feb 1920 tibial graft (Bangour) Oct 1921 tibial graft (Craig cith)	Success	In the case of the first graft both ends strongly united fracture over upper end occurred July 1921 which did not unite, and necessitated second graft All evidence points to very satisfactory result
July 1920 Tibial graft	Success	Walks without a caliper
Aug 1919 Tibial graft	Success	Able to resume his work as a farmer Does not require to wear a caliper Required small skin graft for necrosis of seared skin
June 1922 tepping operation of humerus	Success	Wound healed Firm union Still under treatment
Mar 1920 one graft of humerus intramedullary tibial peg, and chip from iliac crest	Success	Resumed farming in Canada
Oct 1919 intramedullary peg squaring of fragments	Success	Arm can be fully and strongly abducted to angle 70° Good firm union at site of fracture Radial movements from extension practically normal Flexion at shoulder almost to a right angle Hopes to resume work as a riveter
Feb 1920 intramedullary tibial peg squaring of fragment and chips from iliac crest	Success	Necrosis of iliac chips and consequent sinuses Ultimate result of graft satisfactory Abduction at shoulder to angle 60° flexion almost to a right angle Works on a farm

Continued on next page

SYNOPSIS OF 100 CASES OF UNUNITED FRACTURES DUE TO WAR

CASE	DATE OF WOUND AND WHEN HEALED	CAUSE AND PERIOD OF NON UNION	BONE AND SITE OF NON UNION	PREVIOUS OPERATIONS
Case 69—S D	Sept 1918 Oct 1919	2 in gap	R fibula Upper and middle third	Nil
Case 70—J E	Nov 1918 Mar 1921	Sclerosis 28 months	L femur Lower and middle third	Sequestrectomies
Case 71—D H	April, 1918 July 1918	Sclerosis $1\frac{1}{2}$ in gap 17 months	J humerus Lower third	Nil
Case 72—J A	Sept 1915 Nov 1916	Sclerosis $\frac{1}{2}$ in gap 35 months	L humerus Lower fourth and upper three fourths	Two bone grafts in land and sequestra Also inlay bone graft fractured and had to be removed
Case 73—F McC Figs 232, 234	May, 1915 Dec 1920	Sclerosis and displacement 76 months	R tibia Middle third	Sequestrectomies
Case 74—J R	Feb 1918 May, 1918	24 in gap 17 months	R tibia Middle third	Sequestrectomies
Case 75—N McD	Aug 1917 Sept 1919	Sclerosis and displacement 42 months	R tibia Lower third	Two bone grafts in hospitals which were successful Removal of from graft
Case 76—J G	Sept 1917 April, 1918	Latent sepsis 11 months	R tibia Upper third	Abscess in leg opened
Case 77—W H	Oct 1916 Mar 1917	Lower third of humerus missing complicated by flail elbow 27 months	R humerus Lower third	Transplantation of ten ^d Tibial bone graft in ac ^t hospital which had to be removed
Case 78—R G	Oct 1916 July 1917	Articular surface of humerus absent part of olecranon process absent complicated by flail elbow 28 months	R humerus Lower third	Tibial bone graft in an ^t hospital which had to be removed
Case 79—T W	Aug 1916 Nov 1919	Sclerosis 53 months	R humerus Middle of lower third	Sequestrectomy ^{Set} operation for humerus ^{Set} successful
Case 80—F W	Aug 1917 April 1918	Sclerosis 27 months	R humerus Junction of lower and middle thirds	Sequestrectomies ^{Set} in humerus
Case 81—J C Figs 222, 223	Aug 1918 Mar 1919	Sclerosis and displacement 9 months	R humerus Middle third	Transplantation of te ^t drop wrist
Case 82—J B	April 1917 Nov 1917	Displacement 14 in gap 20 months	R humerus Middle third	Tibial bone graft in hospital removal of owing to sepsis

WOUNDS, WITH END RESULTS OF OPERATIVE TREATMENT—continued

DATE AND TYPE OF OPERATION	RESULT	REMARKS
No operation	—	Operation unnecessary as no disability complained of Employed as a labourer
No operation	Success	Femur first united Mar 1920 refractured by slipping on floor, June 1920 also causing wound to break down Treatment consisted of (1) Extension by Thomas's splint, (2) 'Hammer and dam' (3) Calcium ionization and calcium salts internally (4) Endocrine gland tablets (5) Diathermy (6) Paraffin Fracture started to reunite definitely middle of April 1921, and was firmly united by Nov 1921 Employed as a lawyer
Sept 1919 Epping operation of humerus	Success	Ankylosis of elbow and wrist Bone marrow of humerus similar in appearance to that in local union Employed as a clerk
Aug 1918 Epping operation of humerus	Success	Had fibrous ankylosis of elbow, and required 'transplantation operation for drop wrist Employed as a clerk
Sept 1921 Double tibial graft	Success	Marked recrudescence of latent sepsis notwithstanding this, grafts did not die and strong union resulted
July 1919 Tibial graft	Success	Graft was fixed by wire Employed as a miner
Feb 1921 Double tibial graft	Success	Was discharged to out patient treatment and allowed to bear weight on leg too early with partial fracture of one graft Slight recrudescence of latent sepsis occurred after graft operation This did not affect ultimate strong union Employed as a fisherman
United without operation Aug 1918	—	Fracture was transverse with very slight loss of bone By means of Bier's congestion and 'hammer and dam' treatment, good union resulted Employed as a miner
Jan 1919 Sawing of humerus, radius, and ulna, in reduction of chips from iliac crest	Partial success	Increased control of flail joint, and with aid of splint patient is able to work as an electrical engineer
Feb 1919 Arthrodesis of elbow	Success	Able to work as a labourer Has several degrees strong flexion and extension of elbow
Jan 1921 Intramedullary tibial graft, lateral tibial graft and chips from iliac crest	Partial success	Absorption of intramedullary graft union of lateral graft to lower end of humerus, non union of graft to upper end due to recrudescence of latent sepsis Latent sepsis still present
Nov 1919 Stepping operation for humerus	Failure Diabetes	No union resulted Ankylosis of elbow General health unsatisfactory owing to diabetes Marked osteoporosis of lower end of humerus, which fractured when stepping operation was carried out
May, 1919 Stepping operation for humerus	Success	Elbow range of movement from angle 160° to angle 170° Resumed pre war occupation as a postman
Sept 1919 Stepping operation of humerus and removal of wire	Success	Elbow range of movement between angle 150° and angle 90°

Continued on next page

SYNOPSIS OF 100 CASES OF UNUNITED FRACTURES DUE TO

CASE	DATE OF WOUND AND WHEN HEALED	CAUSE AND PERIOD OF NON UNION	BONE AND SITE OF NON UNION	PREVIOUS OPERATION
Case 83—G T	Aug 1916 Sept 1917	Sclerosis 38 months	R humerus Junction of lower and middle thirds	Nine operations for removal of dead bone and metal plate. Squaring and sawing of humerus and fixation with aluminium wire which was subsequently to be removed.
Case 84—A N	Mar 1918 May, 1919	1½ in gap Displacement 24 months	R humerus Junction of lower and middle thirds	Sequestrectomies. Tibial transplantation for drop wrist.
Case 85—T S C	Oct 1918 Nov 1918	Sclerosis and latent sepsis 19 months	L femur Junction lower and middle thirds	Nil
Case 86—J B C	Sept 1918 Jan 1919	— 20 months	P radius Through middle third	Tibial bone graft which had been applied with fixation of host bone not in perfect alignment
Case 87—J V Fig 227	April, 1918 April, 1920	Sclerosis ½ in gap 42 months	R humerus Surgical neck	Sequestrectomies
Case 88—G B	Sept 1918 Oct 1919	Whole of shaft missing 43 months	L humerus Lower two fifths	Sequestrectomies. Tibial graft in other hospital which was ultimately removed due to sepsis. Several skin operations
Case 89—W McD	Oct 1918 Aug 1919	1½ in gap Displacement 40 months	R humerus Junction lower and middle thirds	Nil
Case 90—J H	Aug 1918 Feb 1919	2 in gap Sclerosis 45 months	R ulna Junction lower and middle thirds	Freeing of ulnar nerve, lengthening of flexor tendons for contracted finger. Excision of scar
Case 91—T T	April, 1915 Dec 1915	Displacement and sclerosis 85 months	R humerus Junction of lower and middle thirds	Transplantation of tendon
Case 92—J H	July, 1916 Mar 1918	1½ in gap	L ulna Junction lower and middle thirds	Freeing of ulnar nerve
Case 93—T B Figs 230 231	Aug 1918 May, 1919	Sclerosis and sepsis 19 months	L humerus Lower and middle thirds	Nil
Case 94—W H Figs 228, 229	April 1918 Feb 1919	½ in gap Sclerosis 27 months	L humerus Junction of lower fourth and upper three fourths	Suture of musculospiral Nerve. Wire operation. Bone transplantation
Case 95—J McK	Feb 1918 (Accident)	Ununited fracture of styloid process of ulna 22 months	L ulna Styloid process	Nil

TABLE I. INJURIES, WITH END RESULTS OF OPERATIVE TREATMENT—continued

DATE AND TYPE OF OPERATION	RESULT	REMARKS
Oct 1919 pping operation for for humerus	Success	On examination three years later a very satisfactory result was found. Employed as a haulage engineer in the mines. Finds no disability from his arm. Ankylosis of elbow.
Mar 1920 Tibial graft	Success	Strong union with good alignment.
May 1920 Intramedullary tibial raft, chips from tibia crest, and ring	Success	Wells without a caliper.
Mar, 1920 Previous graft re- moved and new tibial raft inserted with radius fragments in proper alignment	Success	First graft had been inserted with upper fragment of radius completely supinated and lower fragment completely pronated so that forearm movements reduced to nil. By means of fresh graft and correction of alignment good forearm movements obtained.
Oct 1921 stepping operation	Success	Abduction of shoulder to angle 70° 10° of external and internal rotation. Elbow completely ankylosed angle 120°. Forearm fixed in mid position. Strong union resulted. He is satisfied that he can return to his pre-war occupation on the railway.
April, 1922 Double tibial graft fixed by Parham's metallic bands	Success	Still under treatment. Most violent recrudescence of latent infection occurred in the axilla and left flank. Operation scar and graft bed remained intact.
Feb 1922 Stepping operation for humerus fixed by Parham's metallic bands	Success	Still under treatment. Ankylosis of elbow made stepping operation more difficult. Strong union has resulted.
May 1922 Tibial graft	Success	Still under treatment.
May 1922 Stepping operation for humerus fixed by Parham's metallic bands	Success	Still under treatment.
Graft operation could not be carried out as patient had to return to Canada.		
Mar 1920 Intramedullary tibial raft with chips from tibia crest	Success	Elbow movement from angle 160° to angle 80°. This case is remarkable for the large amount of callus thrown out after bone graft operation. Strong union has resulted. Resumed farming work in Canada.
July 1920 Double tibial intra- medullary and lateral one graft	Success	Elbow range of movement from angle 170° to a right angle. Complete absorption of intramedullary graft, although lateral graft fractured partly. Very strong union resulted. Training in commercial work.
Dec 1919 Graft of ulna fixing of lax cap- sular ligament	Success	Employed as a commercial traveller. This operation was performed for recurrent anterior dislocation of the lower end of the ulna complicated by ununited fracture of the styloid process of the ulna.

Continued on next page

SYNOPSIS OF 100 CASES OF UNUNITED FRACTURES DUE TO WAR

CASE	DATE OF WOUND AND WHEN HEALED	CAUSE AND PERIOD OF NON UNION	POS. AND SITE OF NON UNION	PREVIOUS OPERATIONS
Case 96—J H	Mar 1917 Jan 1918	1½ in gap 37 months	R ulna Junction of lower and middle thirds	Nil
Case 97—C S	Sept 1918 May, 1919	1½ in gap 44 months	L ulna Through upper third	Sequestrectomies Suture of ulnar nerve
Case 98—A C	May, 1915 Nov 1917	2 in gap 85 months	L ulna Lower third	Sequestrectomies
Case 99—G M	June, 1917 Mar 1919	1½ in gap 60 months	I ulna Lower third	Plating of ulna
Case 100—C A	Jan 1920 (Propeller accident)	Displacement 3 months	R styloid process of ulna complicated by fibrous union junction lower and middle thirds of radius, and anterior dislocation lower end of ulna	Attempted reduction under anæsthetic in another ho pital, failure

INJURIES, WITH END-RESULTS OF OPERATIVE TREATMENT—*continued*

DATE AND TYPE OF OPERATION	RESULT	REMARKS
April 1920 Tibial graft	Success	Forearm range of movement between complete supination and complete pronation
May 1922 Tibial graft	Success	Marked impairment of pronation and supination
June 1922 Tibial graft	Success	Still under treatment
June, 1922 Fibial graft	Success	Still under treatment
April, 1920 Removal of styloid process of ulna Reduction of dislocation Excision of fibrous union of radius	Success	Remained in army

SHORT NOTES OF RARE OR OBSCURE CASES

METASTATIC MELANOMA OF SCAPULA

B. F. D. CAIRNS GIBRALTAR

THE following case seems worthy of report in view of the very prolonged interval between the appearance of a primary growth and its subsequent dissemination. Eighteen years' freedom from dissemination would be regarded as a remarkable interval in tumours of the most slender claim to malignant character, so that occurring as it did in a melanotic growth of the eye—a tumour usually regarded as highly virulent in its type—that interval is doubly noteworthy. Further, the site of the metastatic deposit is a somewhat unusual one, namely in the scapula, whose immunity from secondary growths was noted by Von Recklinghausen in his researches on metastasis in bone.

G. J., age 59, insurance agent. The patient was admitted under the care of Professor Alexis Thomson for a swelling over the right shoulder-blade. Attention was first called to the condition five months ago, when his friends remarked that he was becoming round-shouldered, an appearance which had progressed gradually, whilst the movements of his arm had latterly become impaired. The patient was conscious of no circumstance which could be held responsible for the appearance of the swelling, and beyond the altered shape of his shoulder and the restricted range of movements at the shoulder joint, he had no complaint.

Eighteen years ago he suffered from loss of vision in his left eye. Examination at that time showed this to be due to a tumour in the eye, and this was removed by Sir George Berry. During the last twelve months he has had varying degrees of difficulty in retaining the artificial eye in the socket, the latter apparently beginning to fill up, and for the last four weeks it has been quite impossible to retain the artificial eye in position. His general health during the last eighteen years has been entirely satisfactory, and his family history does not provide any relevant fact.

LOCAL EXAMINATION.—The swelling was rounded, the size and shape of a lemon, firm and elastic, projecting from and fixed to the infrascapular portion of the right scapula. It moved with scapular movements, and the skin was movable over it. It was neither the seat of pain, nor was it tender to touch. There was no distention of the overlying veins, nor were the axillary glands palpably enlarged. Movements at the shoulder-joint were performed painlessly, but were restricted in range, particularly as regards abduction, by the bulk of the swelling. The tissues occupying the socket of the left eye were unduly prominent, and palpation showed them to be the seat of a firm elastic growth, presumably a local recurrence of his previous tumour. General examination showed no further signs of swellings, and the liver was apparently normal in size and function. The urine was pale in colour and free from albumin and melanin.

OPERATION.—This was performed by Professor Thomson, and the scapula removed. Initially the subscapular artery was exposed by an axillary incision and ligated as an aid in dealing with the hæmorrhage during the subsequent proceedings. The size of the artery in this case, however, hardly warranted the expenditure of time involved, and it is doubtful if the excision of the bone was in any way facilitated by it. It is open to question, therefore, whether or not the preliminary ligation of the subscapular trunk is to be recommended, as suggested by Watson Cheyne.

APPEARANCE OF TUMOUR—The lower half of the infraspinous fossa presents a rounded tumour the size of an orange (*Fig 242*) It does not extend up to the axillary border, but is continuous round the vertebral border, with a flattened nodular extension which lies in the corresponding position of the venter scapula. Firm in consistence, it is mottled in colour, black areas intermingling with white. The bone between these two segments of tumour is destroyed and its place taken by tumour tissue, which effects continuity between them. Section shows the character of a melanotic tumour, bluish-black areas scattered through a cerebriform matrix.

MICROSCOPIC EXAMINATION—The field is almost wholly cellular, though here and there bands of young connective tissue traverse it and divide it into more or less separate areas. Pigment, varying in colour from golden brown where it is scanty in amount to ink black where it is abundant, is scattered irregularly through the section. The bands of connective tissue have a more liberal supply of pigment than the more cellular areas. The cellular content falls roughly into three fields: (1) Areas of cells definitely spindle shaped running in bundles or loosely separated; (2) Round cells loosely approximate with no apparent ground-work, immature blood-vessels, and the characters



FIG. 242.—Scapula with metastatic melanoma.

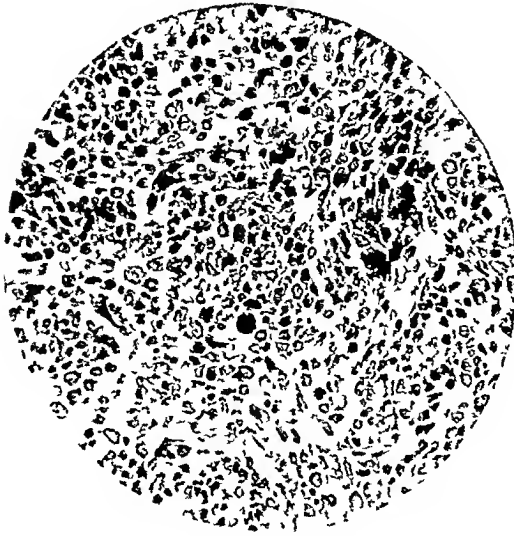


FIG. 243.—Metastatic melanoma of scapula. Area of small round cell.

of a small round-cell sarcoma, (3) Groups of rounded cells massed together in an alveolar-like arrangement, and contained by an investment of large spindle shaped cells (*Fig 243*).

The fact that the young septa are richer in their supply of pigment than other areas suggests that the more mature the cell, the greater its faculty for the manufacture of colouring matter. The spindle cells are of all types, varying from the frank cell to the attenuated unit disappearing in the fibres of a con-

nective tissue bundle. The cell protoplasm stains faintly and the nucleus is prominent, ovoid and clear. Mitotic figures are common. In many cells golden-brown particles of melanin are to be seen, either limited to the nuclear poles or occupying and obscuring

the whole cell body. Blood-vessels are immature and spaced midst the bundles of cells. Round cells of all variations in size occur in groups presenting the features of a round-celled sarcoma. In many cases these cells are multinucleated and show active mitosis. Pigment occurs to some extent in the cells of these particular areas, but is less abundant than in the areas of spindle-shaped cells.

The groups of cells massed together into an alveolar like arrangement are practically free from pigment. The cells are small and rounded and, apart from their massed appearance, do not resemble the spheroidal cells which compose the tumours commonly regarded as melanocarcinomata.

PATHOLOGY.—Both to the naked eye and under the microscope the tumour displays the characteristics of a melanotic sarcoma which clinically and pathologically is secondary to the recurrence in the eye. The pathological classification is that of a mixed-cell melanotic sarcoma, but the occurrence of groups of cells in an alveolar-like arrangement may not be without significance in pointing to the genetically identical relationship which is claimed by some authorities for melanocarcinoma and melanoma. Melanotic tumours arising in relation to the skin, from pigmented moles, or otherwise, have the property of alveolar arrangement of spheroidal cells, whilst they metastasize by the lymphatics. It is found, however, that the metastases show less and less of the alveolar arrangement, and the cells gradually approach one of the types of sarcoma. Nor are metastases confined to the lymphatics, though initially so, and it may well be that lymphatic spread in these so called melanotic carcinomata is determined by their superficial origin.

Ribbert (1897) states that all melanotic tumours have a common origin, be their starting-point the skin, mole, eye, or other pigmented area, namely, in a cell mesoblastic in origin, differentiated from the fibrous tissue cell and existing as a producer of melanin pigment. Such cells he termed chromatophores.

The different cellular arrangements and characters which distinguish melanotic tumours he related to the degree of maturity possessed by the parent chromatophore. Thus the tumour arising from the immature chromatophore of the congenital pigmented mole was more likely to be spheroidal and alveolar in character than the melanoma having its origin in the mature chromatophore of the skin or uveal tract. A recent study of the histology of a series of melanomata of the skin by Hertzler and Gibson bears out this assertion. Ribbert further held that, though the chromatophore is mesoblastic in origin, it was pathologically incorrect to define its tumours as sarcomatous, for like the endothelioma it was worthy of a special designation, and such he found in the word 'melanoblastoma'.

The most striking feature of the foregoing history is the long interval between the original incidence of the primary eye tumour and the subsequent metastasis. The assumption is natural that the scapular growth is the result of dissemination of the recurrence which has gradually manifested itself over the last twelve months, but that in no way detracts from the interest attached to his eighteen years' freedom from affliction. It is authoritatively stated that the expectation of life in melanomata of the eye is rarely more than three years (Blind Sutton). Cases are, however, reported in which dissemination has been as long delayed as eleven years. The interval described in the present case is apparently unique, whether applied to local recurrence or dissemination.

The dissemination of melanosarcomata, taking place as it does by the blood vessels, is body wide, each and every tissue being traversed by the invader. It is exceptional under these circumstances, therefore, for a metastasis to be single and sufficiently dominant in its exhibition to warrant treatment directed towards its removal. Careful examination, however, failed to reveal any symptom or sign of further metastatic growth, though complete x-ray examination might have demonstrated osseous foci elsewhere.

The destruction of bone and its replacement by tumour forms a feature of note, providing a contrast between tumours of the scapula which are primary, and thus which is metastatic.

Examination of the tumours of the scapula in the University Surgical Museum and in the Royal College of Surgeons (Ed.) Museum shows that these are all examples of primary tumours. Their origin is in each case superficial or periosteal, and growth takes place freely over the surface of the bone. The shape and form of the tumour is defined by the muscles which pass over it, whilst they may or may not be invaded according to the pathological nature of the neoplasm. Continuity with a similar tumour on the other side of the scapula is effective by extension round one or other scapular border. Invasion and gross destruction of bone is not a feature of the progress of the tumour. Secondary tumours of the scapula are rare, and whilst Syme in his original commentary on the operation for the removal of the scapula refers to two cases of metastatic growth in the scapula, they were tumours spreading to the scapula by direct continuity, and not embolic.

The invasion and destruction of bone manifest in the present specimen is explained by the medullary origin of the metastasis. Destruction has been too rapid to have allowed of the expansion of the scapula, as may be seen in central tumours of long bones, whilst, once free from the osseous confines, the tumour has grown unrestrained along the surface of the bone on both ventral and dorsal aspects. This central origin is of course in accordance with the embolic origin of the tumour, for the slowed stream of the comparatively widened osseous blood-spaces provides an opportunity for the neoplastic cells to find a foothold.

Secondary tumours in bone are as a rule cancerous in origin. Sarcomatous metastases occur in bone with far less frequency. Melanotic tumours are not covered by this generalization and frequently reproduce themselves in osseous tissue. There is, however, a definite order of frequency in which the bones are affected, and the vertebræ, femur, ribs, sternum, humerus, and cranial bones are, in the above order, the most usual locations of secondary growths.

The exhaustive inquiries of Von Recklinghausen as to the liability of the different bones to metastasis does not lead him to refer to the scapula at all, so rarely is it the seat of such a growth.

The factors which are held to determine the incidence of secondary tumours in bone are those of sudden strain or stress with corresponding alteration in the blood-current in the medullary spaces and in practice the bones most frequently suffering are those subject to the above conditions. The scapula, however, may be considered to live, as far as bones go, a placid, protected existence, and no special circumstances which would explain the unusual incidence, such as occupation or trauma, could be detected in this case.

I am indebted to Professor Alexis Thomson for permission to publish this case.

A CASE OF GUMMATOUS PANCREATITIS WITH PHYSICAL SIGNS RESEMBLING ACUTE CHOLECYSTITIS

By PHILIP H. MITCHNER, LONDON

The patient, J. K., a married woman, age 48, was admitted to the Royal Northern Hospital with a twenty-four hours history of acute abdominal pain and vomiting.

The previous history of the patient was that up to November, 1921, she had been quite well, since that time she had suffered from attacks of abdominal pain, which started in the epigastrium and then travelled to the right scapular region, with accompanying vomiting. These attacks were becoming more frequent and severe. No definite history of indigestion was forthcoming, but the patient had been jaundiced, following one or two of the attacks of pain. The patient has one child, age 9 years, and has had one miscarriage since, her husband is alive and in good health.

The present attack, which was by far the most severe yet experienced, commenced some twenty hours before admission with intense epigastric pain, which radiated to the scapular region and was accompanied by severe vomiting. The bowels had not been opened for two days, nor had anything abnormal been observed in the stools.

On examination a well-nourished but anæmic woman, with slight jaundice. Pulse 104, rapid and of poor volume. Temperature 97.6° (reported by doctor to have been 102.4° earlier in the day). Tongue dry and coated, breath foul. The woman was collapsed and evidently in great pain.

The abdomen was not moving on respiration, and the epigastrium retracted. There was marked muscular rigidity on the right side, especially in the hypochondrium. Palpation in this region elicited extreme tenderness, which was greater on deep palpation. The abdominal reflexes were absent on the right side. Peristalsis could be heard all over the abdomen. A mass could be detected in the right hypochondrium, coming down from under the costal margin. It was indefinite in outline, fixed and tender to the touch. No movement was detected on respiration. Examination of the chest showed diminished air entry at the right base, it was otherwise negative.

In view of the situation of the pain, abdominal signs, and previous history, a diagnosis of acute cholecystitis with perforation was made, and laparotomy was performed by a subcostal incision on the right side.

The gall-bladder presented and was seen to be normal. There was a little free fluid in the peritoneal cavity. A stony-hard mass about the size of a fist was felt in the head of the pancreas, sharply limited below and to the right, but extending up behind the pyloric end of the stomach, and joining with a large fixed mass in the gastrohepatic omentum. This at first sight suggested carcinoma, but further investigation showed it was not fixed to the posterior abdominal parietes, duodenum, or stomach wall; there was, however, distinct constriction of the pyloric end of the stomach due to fibrosis in the mass in this situation.

The omental mass was incised, when about a drachm of blood-stained grumous material escaped, there having apparently been a recent hemorrhage in this situation. A portion of the mass was removed for microscopy, and the peritoneum sutured. The abdomen was then closed and the wall sutured in layers.

A diagnosis of gummatous disease was made and in consequence the blood taken for a Wassermann reaction. The patient ceased vomiting, and made an uninterrupted recovery. The Wassermann reaction was very strongly positive.

The microscopic report on the tissue removed furnished by Dr Shaw, Director of Clinical Pathology to Royal Northern Hospital, was as follows: A mass of omentum and fibrofatty tissue, showing extensive chronic inflammation, and round celled infiltration, probably gummatous.

The points of interest in this case in regard to diagnosis, are —

- 1 The diagnosis of perforation of an acutely-inflamed gall bladder, which was assuredly justified on the physical signs. The subnormal temperature and collapse were taken to indicate recent perforation, and so the absence of peritonitis—as shown by the fact that normal peristalsis was heard all over the abdomen—was regarded as due to its not having had time to develop.

- 2 The possibility, from a superficial examination of the hard craggy mass in the head of the pancreas, of making a diagnosis of carcinoma of that organ and thus of giving a hopeless prognosis. Only a more careful examination showed that the mass was extending in one direction only, i.e., upwards, and did not seem to infiltrate the surrounding tissues in all directions, and tended to negative this diagnosis, as did also the normal appearance of the gall-bladder and bile-ducts, carcinoma could not however, be definitely put out of court on a naked-eye examination alone. Microscopy and the Wassermann reaction were needed in order to make the diagnosis clear.

- 3 The vomiting and pain. These were due apparently to temporary pyloric obstruction from pressure caused by the breaking down of gummatous material, and the consequent stretching of the peritoneum over the mass, with pressure on the pyloric end of the stomach.

A CASE OF CALCIFIED GLAND OF UNUSUAL SIZE GIVING RISE TO DYSPHAGIA

By C P G WAKELEY, LONDON

THE patient, a man, age 54, was admitted to King's College Hospital in May 1922, complaining of a lump in his abdomen. This lump was first noticed about a year previously, and gave rise to no pain until three months before admission when the patient first noticed that at times he had difficulty in swallowing solid food. Liquid foods were easily taken and gave rise to no trouble. It was the dysphagia and loss of weight which brought the patient to hospital.

ON EXAMINATION—The patient was somewhat emaciated and was suffering from anemia. A report of his blood examination was as follows: Red corpuscles, 4,104,000



FIG. 241.—Radiogram taken twenty minutes after a barium meal.

per c mm, or 82 per cent of normal, hæmoglobin, 65 per cent of normal, colour index, 0.8, leucocytes 14,000 per c mm. The urine was normal. Wassermann reaction was negative. On examination of the abdomen a hard solid tumour was felt to the left side of the epigastrium. It was about the size of an orange, and could be easily moved about the abdomen. It could not be felt on examination per rectum. A full-size œsophageal bougie was easily passed demonstrating no obstruction in the œsophagus.

An opaque barium meal was given to the patient, and a radiogram taken twenty minutes afterwards (Fig. 244). The stomach was of normal size and shape, and a duodenal cap was just beginning to form. A large, somewhat circular opacity of about three inches diameter was seen lying over the promontory of the œsophagus. Subsequent skiagrams

proved the intestinal tract to be normal from the radiographic standpoint. *Fig 245* is a radiogram taken twenty-four hours after the ingestion of the opaque meal; it shows the barium in the cæcum, ascending and transverse colon, and splenic flexure. The opaque rounded body can be easily seen.

Six days after the barium meal another skiagram was taken with the patient in the upright position. The opaque body was then seen to be overhanging the brim of the pelvis.

The diagnosis seemed to rest between a calcified dermoid cyst and an enormous calcareous gland in the mesentery.



FIG 245.—Radiogram taken twenty four hours after a barium meal

A laparotomy was performed by Mr Burghard, in May, 1922. Under the anaesthetic the lump could easily be moved diagonally across the abdomen in the line of the mesentery.

On opening the abdomen through a left rectus incision, a large calcareous mass in the mesentery was delivered, this was carefully dissected out of the mesentery by incising the upper leaf of the membrane, great care was taken, because the branches of the superior mesenteric artery and vein were stretched out over the mass, and a few had to be divided between ligatures. After removal of the tumour, the peritoneum was carefully sutured and the vitality of the gut was not endangered. The abdominal wound was closed in layers. The gland, which was almost calcified throughout, and is shown in reduced form in *Fig 246*, measured three inches across, about the size of a large orange.



FIG 246.—From a photograph of the gland ($\times 4$)

The patient left hospital after two weeks, he could eat anything, and he has been seen since and has gained almost a stone in weight. This case is of interest because of the unusual size of the calcified gland, and its pressure effect on the stomach.

UNUSUAL COMPLICATIONS IN TWO CASES OF
FEMORAL HERNIA

By S. LAWRENCE LUDBROOK, NEW ZEALAND

1 ACUTE APPENDICITIS IN THE SAC OF A FEMORAL HERNIA

THE incarceration of the vermiform appendix in the sac of a right femoral hernia is a well-recognized surgical possibility and the appendix may of course become inflamed whilst in this abnormal position, indeed, it must be especially liable to do so by reason of the necessary interference with its blood-supply and stagnation of its contents. The following case illustrates the sequence of events, and serves to emphasize the great difficulty presented in diagnosis. For these reasons it is thought worthy of a brief record.

H. C., a man, age 36, was admitted to hospital April 19, 1922, complaining of a painful swelling in the right groin.

HISTORY OF ILLNESS—The patient was an exceptionally healthy man until six months previously, when he noticed a small swelling in his right groin. Soon after it first appeared it quite suddenly began to increase rapidly in size, and became red and painful. He consulted a medical man, who diagnosed tuberculous abscess from broken-down lymph glands. The condition was treated by aspiration, when a quantity of sero-pus was removed. This relieved the pain, and the patient was able to resume work.

Three months later he had a sudden attack of very intense pain radiating from the umbilicus downwards and laterally into the right iliac fossa and the thigh. The acute pain lasted eighteen hours, and was followed by a soreness lasting several days. A few hours after the pain had ceased the swelling became enlarged and tender. After recovering from this attack he carried on his work for another six weeks, when he had another attack very similar in character, but not so intense. It was after this last attack had quietened down that he was admitted to hospital.

During the six months he had lost a good deal of weight, and had been troubled with loss of appetite and constipation.

EXAMINATION—On admission, temperature was 99°, and pulse 72. The patient was thin, and not complaining of any pain. A hard nodular mass was found in Scarpa's triangle on the right side. The overlying skin was red, slightly oedematous and seemed to be attached to the mass. No impulse could be felt on coughing, and no fluctuation elicited. The mass was quite irreducible.

DIAGNOSIS—A diagnosis of femoral hernia was made with a good deal of uncertainty, and an operation was performed on April 26.

OPERATION—An oblique 'inguinal' incision, curving vertically downwards at its inner end, was made, and a flap thus turned outwards, some difficulty being experienced in separating the adherent skin from the thickened mass of inflamed glands, which formed the more superficial part of the mass. Some of the glands contained thick yellow pus. By dissecting round the upper part of the mass the greatly thickened hernial sac was discovered.

On opening into the sac it was found to contain the appendix, acutely inflamed, and firmly attached to the end of the sac. The opening into the abdomen was obliterated by adhesions to the neck. The inguinal wound was temporarily packed off, and the abdomen opened by a separate pararectal incision. The caecum was brought into the wound, and the appendix removed at its junction with the caecum, the stump being buried in the usual manner. A ligature was applied to the cut end of the appendix, the hernial sac was cut through at the neck, and the stump of the appendix everted through the sac. The lymph glands, thickened sac, and appendix were then removed *en masse*. The femoral canal was repaired, and the wound completely closed.

Healing took place by first intention, the patient being discharged on May 2 to a convalescent home.

The photographs (Figs 247, 248) show both aspects of the specimen the appendix lying in the enormously thickened sac, and adherent at its tip to the fundus of the sac. The outside of the sac fundus is covered by a mass of adherent lymph glands.



Figs 247, 248—Show the appendix in the sac of the hernia both aspects
A, Appendix S Sac wall with glands adherent

2 A CASE OF RIGHT FEMORAL HERNIA COMPLICATED BY A MECKEL'S DIVERTICULUM ADHERENT TO THE HERNIAL SAC

THIS case is of interest as compared with the above one of appendicitis occurring in the sac of a right femoral hernia, and also as an example of one of the more unusual complications of abdominal hernia more especially of the right side.

F. E., a man, age 52, was admitted to hospital on May 21, 1922, complaining of a painful swelling in the right groin.

HISTORY—Eight years previously the patient developed a swelling in the right groin. This was reduced and after wearing a truss for some years it disappeared completely and caused no further symptoms until a fortnight before admission, when it reappeared. Four days later the swelling became painful, and he found that he could not reduce it.

EXAMINATION—This disclosed a soft swelling on the right side below Poupert's ligament, and below and external to the pubic spine. The swelling was quite soft, gurgled on manipulation, and a definite impulse was felt on coughing, but the bowel contents of the hernia could not be reduced.

OPERATION—An oblique inguinal incision was made, extending vertically downwards at the inner end into the groin. A flap was turned outwards, and the hernial sac isolated from the subcutaneous tissues and incised. The incision opened bowel, which on further investigation proved to be the thin walled apex of a Meckel's diverticulum adherent to the sac. The diverticulum was closed and, after careful dissection, separated from the sac and returned to the abdomen. The abdomen was then opened by a right para-rectal incision, the diverticulum clamped and removed, and the bowel wall sewn over. The hernial sac was then dealt with in the usual manner.

Both these cases were operated upon by Mr T. Twistington Higgins and I am indebted to him for permission to publish them.

ULCERATION OF THE RECTUM, WITH PERFORATION INTO THE PELVIC CAVITY, AND PROLAPSE OF ILEUM PER ANUM.

By CYRIL H CUFF, ST LUCIA B W I

THE patient, a West Indian man, age 30, was admitted to the Victoria Hospital, Castries, Feb 3, 1922

HISTORY—In November, 1921, the patient came into hospital complaining of difficulty of micturition. He had a stricture which admitted a No 8 bougie. He refused to stay for treatment, and left on the following day.

ON RE-ADMISSION Feb 3, 1922—The patient presented an extraordinary appearance. He was lying on his face vomiting copiously, and protruding from his anus were coils of distended small intestine, about four feet in length, and of a dark-blue colour (*Fig 249*). The temperature was 97° pulse 70, the patient cold and collapsed. He stated that sixteen

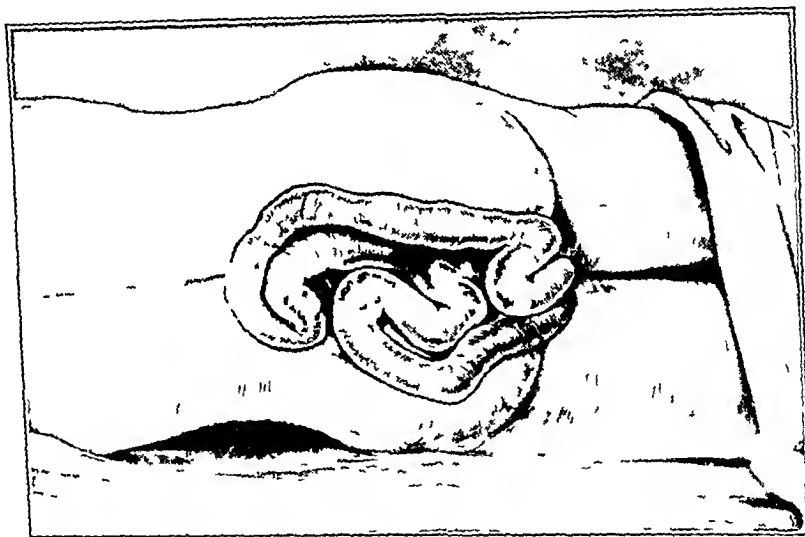


FIG. 249—Showing the condition of the prolapsed portion of the ileum

(The illustrations are all reproduced from the original sketches.)

hours previously whilst straining to pass urine "he felt something go inside" and experienced a peculiar sensation about the anus, and a "sinking feeling" in the abdomen. He then became aware of something moist and warm between his thighs, and on inspection found several inches of intestine prolapsed. He was far away in the bush at the time, and sent for assistance. Meanwhile the desire to micturate still being present and the strain continuing more bowel descended and in due course began to distend. He then began to vomit and experienced severe abdominal pain, especially in the region of the umbilicus. Help arriving he was carried twelve miles in a hammock to hospital. Obviously there was some abnormal communication between the rectum and the peritoneal cavity and the prolapsed gut was either constricted thereat, or twisted upon itself. It was decided to attempt a reduction as offering the only chance of recovery, and a preliminary intravenous saline was administered.

OPERATION—Spinal analgesia (stovaine) being induced and with the patient in the lithotomy position the prolapsed bowel was thoroughly washed with warm saline and covered with hot towels. The abdomen was then opened in the middle line below the umbilicus,

and the intestines carefully packed off. It was now seen that a loop of ileum, about six feet from the ileocaecal valve, led down to the right pararectal fossa, where it disappeared through an opening in the peritoneum and side of the rectum, just above the lateral reflexion. Gentle pressure was now applied from below, while the bowel above was carefully manipulated. The constriction was found to be slight, but the prolapsed bowel was twisted about three-quarters of a circle upon itself. The volvulus (a secondary one) was corrected and the bowel returned without difficulty. The general appearance of the gut improved considerably and it was decided to leave it in the abdomen. A long gauze drain was passed through the opening from the pelvis and out at the anus. The patient being extremely collapsed, the wound was closed with through-and-through sutures, and a further saline with pituitrin given.

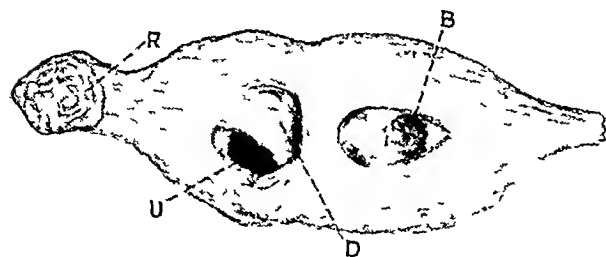


FIG 250—Superior view of the specimen (R) Rectum (B) Bladder (U) Site of perforation (D) Pouch of Douglas

—At the post-mortem, the whole of the rectum, bladder, and pelvic peritoneum were removed *en bloc* and the bowel slit open from behind. The mucous membrane showed several small ragged ulcers, varying from minute spots to the size of a shilling. The edges were irregular, and the floor rough and covered with mucus. There were numerous petechial hemorrhages. The site of perforation was situated about $3\frac{1}{2}$ in from the anus, on the right lateral wall. It measured about 1 in by $\frac{3}{4}$ in, and was roughly circular. The edges were rough, somewhat undermined, and rather hard. The overlying peritoneum was faintly adherent and, where perforated, was white and fibrous, with jagged edges (Figs 250, 251).

The muscular coat of the bowel around this area was much thickened. The impression gained from an inspection of the specimen was that the floor of the ulcer had for some considerable time consisted solely of the thickened peritoneum and that this had given way suddenly under the constant strain at micturition. There was no sign of ulceration elsewhere in the gastro intestinal tract. The ulceration was probably of syphilitic origin, syphilis being not uncommon here. Microscopically there was merely a dense round-cell infiltration and fibrous overgrowth.

Bilharzia does not occur in St. Lucia.

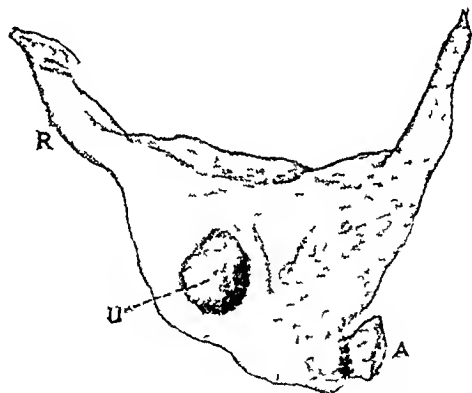


FIG 251—Lateral view of the specimen (R) Rectum, (U) Site of perforation (A) Anal

I am indebted to Dr H. G. Sutherland Richards, M.C., for the sketch of Fig. 249, and to Mr. Donald Devaux for Figs. 250 and 251.

TORSION OF THE GALL-BLADDER

By C H S FRANKAU, LONDON

Torsion of the gall-bladder is sufficiently rare to justify the recording of the following case

The patient a woman, age 62, twenty-four hours before admission into hospital complained of abdominal pain which commenced about an hour after her mid-day meal. The pain was mainly in the upper abdomen, and was colicky in nature, in the evening it became more intense, and she sought medical assistance. When seen by her doctor she had a normal temperature and pulse, and presented no abdominal signs apart from some tenderness in the upper abdomen. She was given a sedative and was relieved for a time, but during the night she commenced to vomit, and continued to do so incessantly. She was seen by her doctor the next morning, and immediately sent to hospital. There was no history of any previous abdominal trouble.

ON ADMISSION.—The patient looked ill. Temperature 97.6° , pulse 112, respirations 24. The tongue was dry and furred. The abdomen showed no distention, respiratory movements were almost completely absent. On palpation, there was general tenderness with rigidity, this was most marked in the right upper quadrant, where the rigidity was absolute. The lateral liver dullness was absent. No tumour could be felt, either before or after the induction of anaesthesia.

In spite of the continued vomiting I considered the case to be one of perforation of a gastric or duodenal ulcer, and opened the abdomen through the right upper rectus shortly after her admission. On opening the peritoneum the liver, which was prolapsed, presented, on lifting the liver up, a small quantity of blood stained fluid escaped, and the gall-bladder was seen to be black in colour, oedematous, and somewhat distended. Further investigation showed that the gall-bladder had rotated for one complete turn from right to left, the rotation being primarily on a short mesentery by which it was attached to the liver. The rotation on the mesentery had kinked the gall bladder, so that its medial surface was markedly concave and a partial hour glass constriction had been produced (Fig 252). The mesentery was fan-shaped, being attached for a distance of 18 mm. to the gall-bladder and for about half as far again to the liver, the depth of the mesentery was approximately 30 mm., and its leaves at the hepatic attachment were in apposition. There were no stones in the gall-bladder, and the common duct was clear. The stomach was low in position, the gastrohepatic omentum being abnormally long, the right kidney did not appear to be unusually low or mobile.

The gall bladder was separated from the liver by division of the mesentery, and removed after double ligation of the cystic duct. The abdomen was then closed in layers, a split tube drain being left down to the operation area. Recovery was uneventful.

REMARKS.—The rarity of the condition is dependent on the infrequency with which the gall bladder is completely surrounded by peritoneum and is suspended from the liver by a definite mesentery. A well-formed mesentery is present in about 5 per cent of gall-bladders, and only a small percentage of these being free, since not infrequently a prolongation of the small omentum anchors the fundus to the duodenum or transverse colon.

Rotation in this case took place primarily on the mesentery, the cystic duct being only secondarily involved, this was made possible by the shape of the mesentery and



FIG 252.—Gall bladder, showing the constricting effect caused by the rotation.

by its comparatively short length of attachment to the gall-bladder as compared with its hepatic attachment. In other respects the case conforms with those already published, for details of which, and for the sketch of the removed gall-bladder, I have to thank my friend Mr J A Cairns Forsyth.

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REMOVAL OF A PIN FROM THE THIRD PART OF THE DUODENUM

By E. E. HUGHES, MANCHESTER

THE patient, a girl, age 4 years, was admitted to the children's ward of the Ancoats Hospital on Sept 1, 1921. The history, as given by the mother, was that the child had swallowed a pin. On x-ray examination a large pin was seen lying at the level of the 4th dorsal vertebra on the left side. The child was given porridge in the hope that the pin would be passed naturally. The following day it was seen to occupy the same position as on the previous day. On Sept 3 a radiograph was taken, the child being in the supine

position, and the pin was now seen to be at the level of the intervertebral disc between the 3rd and 4th lumbar vertebrae, and placed obliquely at an angle of 45° (Fig 253). In the afternoon a simple enema was given, with a good result. On Sept 4 the patient looked quite well and complained of no symptoms. Another radiograph was taken, and showed the pin lying in exactly the same position as on the previous day. Operation was decided upon for the following day.

On Sept 5 the abdominal cavity was opened through a right rectus incision. The stomach was first very carefully examined for the pin, with a negative result, and, similarly, the transverse colon. The whole length of the jejunum and ileum was next examined without result. The incision was then slightly enlarged to allow of deeper access, and the duodenum was carefully



FIG 253.—Pin in third part of duodenum

examined. In the third part of the duodenum, about 2 inches from the duodenojejunal flexure, the pin could be felt, its point presenting forwards and upwards to the left. The point was expressed through the intestinal wall, and the pin was pulled through as far as its head would allow. A purse string suture was then run round the pin, which was then extracted by a sharp tug. The orifice so made was immediately closed by the prepared suture, and reinforced by a few Lembert sutures. A long retrocaecal appendix in healthy condition was found, and removed. The abdominal wall

was sutured in layers. The patient bore the operation well but suffered from a certain amount of shock. Rectal salines were given four hourly for twenty four hours and fluids were administered by the mouth. On the following day the condition of the child was much improved and thereafter she made an uninterrupted recovery. The patient left the hospital on Sept. 20 in excellent health and with the wound soundly healed. Seen at intervals since discharge from the hospital the child has continued to enjoy good health. The length of the pin—in ordinary domestic pin—was 31 mm.

I am indebted to Dr. J. M. Morrison, radiographer to the Ancients Hospital for the radiographic print.

PANCREATIC FIBROSIS OBSTRUCTING BOTH THE COMMON BILE-DUCT AND THE DUODENUM FIVE YEARS OF ACTIVE LIFE AFTER CHOLECYSTDUODENOSTOMY AND GASTROJEJUNOSTOMY BEFORE DEATH FROM CANCER.

By W. G. SPENCER, London.

IN 1908 a cavalry officer, age 30, who had served for seven years in South Africa without illness, during the voyage home was seized with acute pain for the first time. After arrival he consulted Drs. Gee and Mitchell Bruce and the diagnosis made was that he had had an attack of gall-stone colic. Dr. Ironside Bruce did not discover anything by perity examination. Obscure attacks of indigestion, without at any rate noticeable jaundice followed, but his general health was not impaired and he continued to play polo for his regiment.

In February, 1912, before the operation he presented two positive signs—slight resistance with tenderness on deep pressure under the right 9th rib cartilage and Cammidge's urine reaction definitely positive. At the operation the head of the pancreas without being enlarged was found densely hard and nodular, it was also compressing both the common bile-duct and duodenum causing some dilatation. In the search for calculi the pancreas was cut into—it was densely hard and grated under the knife like scirrhus cancer. On opening the duodenum no change on the mucous aspect was found—only a compression of the lumen without any sign of a stone at the papilla or elsewhere in the bile-duct. The gall-bladder was unaltered and its contents were normal bile—it was anastomosed with the duodenum above the pancreas and the abdominal wall closed except for a temporary drain. Recovery was complicated by gastric dilatation relieved by washing out.

The dilatation became worse on getting up so three weeks after the first operation, and after a consultation with Sir Rickman Godlee gastrojejunostomy was done, when it was noted that all the body of the pancreas was indurated. The statement made, not to the patient, but to his brother-in-law, was that the disease was cancer. The patient made a rapid recovery, returned to his regiment, and resumed polo. Subsequently he married. On the outbreak of war he went out with his regiment as a major, and was in the retreat from Mons. He continued at the front and was twice wounded, when he had to return temporarily to England. He gained the D.S.O. and the Croix de Guerre, there is no report of any sickness until, in April, 1917, he was taken to hospital suffering from an acute abdominal attack. On exploration of the abdomen, generalized malignant disease was found, and he reached home a few days before his death a little more than five years after the two anastomoses. All that can be said is that the opinion given from observations made at the two operations was that the condition was already one of scirrhus cancer of the pancreas. It may be, however, that cancer supervened late upon a dense fibrosis.

TWO CASES OF RUPTURE OF THE RECTUM, COMMUNICATING WITH THE PERITONEAL CAVITY

By W G SPENCER, LONDON

In the one case the end of the handle of a wheelbarrow, in the other the end of a chair leg, pushing the seat of the breeches in front of it, passed in through the anus and ruptured the anterior wall of the rectum.

Case 1—It was only in the course of the coroner's inquest and of the inquiry held at the Hospital afterwards that the correct story of this accident was made out. When the offices of the Ministries of Health and of Education were in course of building, the man was standing on a wall which had been raised two feet from the ground, when he stepped backwards off the wall against a wheelbarrow. The wheelbarrow had been turned on its side so that the upper handle stood out at an angle of some 45° from the horizontal. A fellow-workman assisted the patient about 100 yards to the Westminster Hospital, where he was seen by an assistant house surgeon, who noticed blood in the seat of the man's breeches and a small abrasion to one side of the anus. He applied an antiseptic dressing to this, and sent the man away. The house surgeon afterwards excused himself for not making any further examination or admitting the case, because the two men had given no clear account of what had happened, and he had mistaken for fright the man's general condition, which must have been due to shock. The man went home and—acute septic peritonitis setting in—he was admitted to the Bolingbroke Hospital, where an immediate operation was performed. A rupture of the rectum into the peritoneal cavity was found, but it was too late to save the patient's life.

Case 2—A boy of 7, at Eastbourne, was playing at what he called 'submarines', in the course of which he had turned up the nursery chairs so that the hinder legs stood out at about 45° from the floor. Against one of these he staggered back and was able to give a sufficient explanation of what had happened to his mother, who found blood in the seat of his knickerbockers. She sent for Dr Harper. There were no external signs of the accident, but there was a rent on the front wall of the rectum, and blood in the urine. Acute septic peritonitis became obvious within six hours of the accident, and we operated within twelve hours. The boy was then suffering from well established septic pelvic peritonitis, the pulse rate was 130 and small, the face pale and pinched, the abdomen already somewhat tympanitic. On making a median hypogastric incision, blood stained septic fluid escaped, and the peritoneum was found inflamed, but no actual rent was detected. The urine contained blood, but the bladder proved watertight.

On dividing the sphincter and backwards to the tip of the coccyx the rent in the rectum was fully exposed. It was situated on the anterior wall just above the internal sphincter, the margins were ragged and infiltrated by blood. Exploration with the finger and probe did not reveal any actual communication with the peritoneal cavity. The high position of the bladder and the formation of the pelvis in the little boy had caused the laceration to extend into the rectovesical fascia, and the actual penetration of the peritoneal cavity must have been a merely valvular puncture, although sufficient to start acute septic peritonitis forthwith.

A consideration of the above features appeared to oppose any attempt at suturing the rectum. As the boy lay on his back there was a conical wound, the base of which was exposed so that a dressing could be applied to it. The blood in the urine could be accounted for by a contusion of the mucous membrane. Therefore the abdominal wall was sutured except for a drainage tube, the inner end of which lay over the bladder which was kept empty by a rubber catheter retained in it. Dressings were applied to the laceration on the anterior wall of the rectum until the anus closed. The peritonitis was arrested, and all healed without complication, so that no trace of the accident remained.

REVIEWS AND NOTICES OF BOOKS

Treatment of Injuries of the Peripheral Spinal Nerves. By Sir HAROLD STILES, K.B.E., F.R.C.S., Regius Professor of Clinical Surgery, University of Edinburgh, and M. I. LEONARD BROWN, M.S., M.D., formerly Surgeon, Edinburgh War Hospital, Roy. Soc. Pp. 180. xvi. Illustrated. 1922. London: Oxford Medical Publications. 15s. net.

IN the introduction to this book, based on a large experience in the treatment of war nerve injuries, the writers set themselves the task "to map out for the surgeon who has no special experience of the subject those paths which will lead to a successful result for himself and his patient, and to help him to avoid those pitfalls which have entrapped most workers at first, before they learned to look out for them." This has been successfully done.

The anatomy of the nerves most commonly injured is well described. There is one small point with which the reviewer disagrees. In *Fig. 3a* depicting the sensory loss after complete division of the musculospiral nerve, an area of loss of epicritic and protopathic sensibility is shown over the terminal phalanx of the thumb on its dorsal aspect. In *Fig. 6b* this same area is given as being insensitive to deep pressure and pain after division of the median nerve. It cannot be usual to find these areas, they may perhaps be accounted for by amputation of branches of other cutaneous nerves in severe war injuries.

The section on diagnosis gives everything necessary in short compass, nothing important being omitted. The chapters on operations are the fullest that have been published and treat the subject in an exceptionally able way, both from the general aspect and also in the description of its application to individual nerves. The volume closes with a clear description of the indications for tendon transplantation in nerve injuries and the methods of performing the operation. This is one of the most valuable chapters in an excellent manual.

The illustrations of operations are good and indicate all the points clearly. Many of the reproductions of photographs, however, are so poor that it is difficult to make out the points they are inserted to show.

The writers are to be congratulated on the production of a book that should be in the hands of all who have to deal with this type of injury.

A Text book of Surgical Anatomy. By WILLIAM FRANCIS CAMMIE, A.B., M.D., F.A.C.S., Surgeon in Chief, Trinity Hospital, New York. Third edition, revised. Medium 8vo. Pp. 861 with 325 illustrations. 1921. Philadelphia and London: W. B. Saunders Co. 30s. net.

FROM the fact that this volume has already gone through two editions, and some fourteen years after its first appearance is still in demand, one can only deduce that it filled a gap in surgical literature, or has created a place for itself. Destructive criticism in a review of a book of this kind is exceedingly easy, yet one cannot but compare the comparatively meagre and not always accurate letterpress of the volume under review with that mine of information in pocket form which we have known throughout our medical career to be *THE* *Surgical Applied Anatomy*. As a make-weight on the other side of the balance, one must admit that with regard to print, paper, illustrations, and general appearance, the volume under review is as much in advance of any British publication of the same kind as American books—particularly those coming from the house of Saunders—usually are. It is the letterpress that we find so poor and disappointing. For instance, we are told that preliminary ligation of the lingual artery is a simple and effectual means of lessening hæmorrhage during excision of the tongue, since there is little or no anastomosis between the two halves of the tongue. Surely it is agreed that this proceeding is only of preventive value if carried out within a few days of the excision, for the very reason which is the negation of that given above. Two paragraphs later follows a list of tumours which it is said are fairly common in the tongue. The value of such a statement is doubtful, even if its accuracy—which is not in agreement with our experience—is admitted.

On p. 171 a diagram shows six cords entering into the brachial plexus, even if there were more than five of classe memory, they could not have the relations here depicted. Similarly a diagram on p. 297, though a beautiful and absolutely true representation of the common supracondylar fracture of the humerus, is wrongly labelled "Separation of the epiphysis at elbow."

It is a pity that there should still be text books of merit which continue to spread the old mistake that this injury is a separation of the epiphysis. In truth of course, the fracture involves the humerus some $\frac{2}{3}$ in above the epiphyseal line, and it is difficult to understand how it can be caused by a fall on the elbow, or by 'jamming' the elbow in a door. It is to be regretted also that on p 308 fixation of the forearm in fracture of both bones should be recommended midway between pronation and supination, though if the author's results are entirely satisfactory, he has been more fortunate than ourselves, and is to be congratulated. The spine and spinal cord are dismissed in thirteen pages, and we find no diagram showing the areas innervated by various segments. The clinical pictures of spinal bifida and fractures of the spine are meagre and anatomical facts connected with the common reflexes are entirely omitted.

While the illustrations are beautifully executed, and the majority serve to assist the reader, some would seem to be scarcely worth including. Those which appear on the pages dealing with hernia do not appear to be accurately drawn, and for a long time we failed to recognize the testicle. On p 549 we are told that "tuberculous affections localize in the head of the epididymis" a diagnostic point which we hoped had been discarded as untrue.

The recent researches of Flint into the anatomical variations of the common bile duct have shown that in an appreciable percentage it is not formed until some distance below the normal level. It is to be doubted, however, if any of the variations demonstrated by Flint show the arrangement depicted on p 431, nor is it helpful to the surgeon to remark that "the supraoduodenal portion of the common bile duct is very short". The figure accompanying this statement is inaccurate in the same direction as the one on p 431, and as the supraoduodenal portion of the common bile duct is the seat of election when surgical intervention is necessary, it would be more helpful to state its average length than to be satisfied with a remark such as that quoted above.

The figure on p 279 depicts a claw hand due to paralysis of the ulnar nerve. Surely such a condition as is here shown cannot result from a lesion of the ulnar nerve only. Similarly, in connection with the musculospiral nerve, it is an omission of considerable clinical importance not to point out that lesions near the elbow lead to non sensory symptoms, and in a book of this size one would expect some real information about the anatomy of Erb's paralysis. On the whole, we very much prefer an old friend which may be carried easily in the pocket.

Collected Papers of the Mayo Clinic Rochester, Minnesota Vol XI 1919 Edited by Mrs M H MELLISH Large 8vo Pp 1331 Illustrated 1920 Philadelphia and London W B Saunders Co

We have thought in the past that the view point of work of the Mayo Clinic was somewhat confined to certain areas of the surgical field, and further that it was limited, out of all fair proportion, to the clinical aspect of disease. If such criticisms were fair and accurate in the past they can certainly no longer be upheld, for the papers composed in this volume range over all parts of the body, and include the fields usually reserved for the specialists. Those which deal with surgery in its clinical aspect—and happily they still occur in a lesser proportion—because they are likely to interest our readers, receive a notice here out of all proportion to their number in the volume, but this must not be assumed to assess their value is high as those of wider or purely scientific interest.

As regards the range of subjects, it can scarcely be extended as it includes on the one hand an article by L B Wilson on *Graduate Medical Education in Great Britain and France* and on the other, *A Note on Scalpel Sharpening*. Having said this the writer must confess that this extension of the field which has to be covered very much increases his difficulties for it is scarcely possible for a reviewer to combine the expediency to assess the value of a purely clinical paper with that which would judge the real value and permanence of papers which deal with purely pathological problems. While in no sense assuming the knowledge fully to appreciate its value or meaning the writer must confess that the first paper or series of papers which attracted his attention were those by Kendall and Osterberg, either alone or in combination, on *The Chemical Identification of Thyroxin and its Physiological Action*. Kendall's paper on the last subject is extraordinarily instructive and suggestive, and it is to be hoped that the line of investigation herein outlined will be pushed further, not only in relation to the influence which the active principle of thyroid has upon the human organism, but also with the object of estimating the individual and collective influence of the secretions of the endocrine glands upon normal and abnormal man.

Irene Sandilord's paper on *The Basal Metabolic Rate in Exophthalmic Goitre* is of the same high level, and of course has the immense advantage assured by all scientific papers from this Institution, in that it is based upon a wealth of clinical material.

One of the most fascinating and disappointing questions which seem recently to have come into increasing prominence in the world of surgery is that of organ transplantation. Kowamura reviews the history of this subject in his paper which will be helpful to anybody contemplating either experimental or clinical work upon this question, for he lays down fairly definitely certain general principles which it is by now generally agreed dominate this question. He may claim to have proved that the thyroid gland and spleen may be transplanted with intact blood supply.

autoplastically but not homoplastically in the dog. It is to be hoped that the work of Carrel and others in the last fifteen years may enable us to see the day when organ transplantation may be a practical problem in man, and in any case those who have an acquaintance with the extraordinarily high level of technique demanded in this kind of experimental surgery, for Carrel noted in 1907 that it required a higher degree of asepsis than ordinary surgical procedures, must ungrudgingly award the experimenter a word of praise for his wonderful work.

In recent years Judd's writings have tended to centre round the urinary tract. His papers on surgery of the kidney and removal of stones from the ureter are sound, replete with clinical insight, and from the volume of experience which they represent would be invaluable to anyone embarking upon this type of surgery. They contain perhaps the most beautiful drawings which we have seen even from the pencil of Miss Fry. This artist's skill in the illustrations of Dr C. H. Mayo's paper on *The Surgical Treatment of Cancer of the Stomach* does much to enhance its value. We assume that the selection of depicting those steps in the operation which appear to be essential and instructive has been in the surgeon's hands, but even then one may be permitted to remark that this selection to us appears to be ideal, and moreover that the illustrations, while artistically perfect, sacrifice nothing of clearness or detail to artistic effect.

Of the several papers on the treatment of the diseases of the duodenum and stomach which we naturally expect to find in any volume of this series none calls for special attention yet all justify their inclusion because they push a stage further some suggestive line of thought, or endorse a principle or practice by showing that a further period of trial has proved its worth.

We do not appear to have come across J. C. Masson's writings in previous volumes, and if it is an oversight on our part the loss is ours, for his short papers on *Exposure in Gall-bladder Surgery* are a model of what the description of an operation should be. J. B. Reeves' paper on *The Inferior Supply of the Stomach and Duodenum* is already widely known. It must represent a colossal piece of work, and the microphotographs of injected specimens have obviously been meticulously made. If he has not established any definite role for the autonomic arrangement of the vessels in the causation of ulcers, his work is at least sufficiently conclusive and exhaustive to induce to others searching for the truth of this question that it is not along these lines that they must seek for the chief factor.

W. J. Mayo's paper on *Results of Splenectomy in the Animas* represents the best which such an aggregation of clinical experience can produce, as from no other one institution in the world can records of 61 splenectomies for splenic injury and 27 for hemolytic icterus be brought together. The full value of such an experience will only be appreciated and that most gratefully by those whose opportunities for such work are few indeed.

Collected Papers of the Mayo Clinic Rochester, Minnesota. Vol. XII, 1920. Edited by Miss M. H. Minnich. Large 8vo. Pp. 192. Illustrated. 1921. Philadelphia and London: W. B. Saunders Co.

The Mayo Clinic has become since the war a vast institution in which all the specialties and associated sciences are represented in the different departments. *Pari passu* with this extension and subdivision of their work, the type of papers comprised in the volume under review tends more than in its forerunners to specialism, either clinical or scientific.

Eusterman contributes a valuable study of 83 gastroduodenal ulcers verified at operation, and concludes that this lesion is largely due to technical error or mechanical defect in the operation. MacCarty adds another of his papers on the *Classification of Neoplasms*. We have tried, from this writer's conception, a working classification which could be understood by the ordinary student, but must confess that the introduction of so many new terms, and such a vast number of subdivisions makes it difficult, and we cannot think that his scheme is likely to meet with general adoption.

Henderson contributes several articles dealing with non-union for which the use of beef bone screws is advocated. The practice is ingenious, but seems to be contrary to the general principles which govern the present practice of bone surgery. It is admitted that their resistance to stress and strain is limited, is heterogeneous absorbible material, it is clear that sooner or later they disappear, but no experimental or clinical evidence is forthcoming to suggest that this absorption does not antedate the true union of and around the graft. After reading these papers, we are at a loss to appreciate what advantages they possess over wire or metal screws, and it would appear that the difficulty of ensuring their sterility is far greater.

Masson reviews the statistics of over 10,000 herniæ, which comprise more than 2000 in the Mayo Clinic during the years 1915-17. He appears to accept the idea that the hernial sac may be either developmental or acquired. It is interesting to have statistical evidence of the success of the Mayo operation for umbilical hernia, for this operation in the hands of those who originated it gives a recurrence of less than 1 per cent. It seems at first sight strange that the Mayo series of 2000 cases should include only just over 100 femoral hernia, and even when we take into account the fact that the period dealt with is that during which men were being rendered fit for military service, the proportion seems smaller than we should have anticipated. It appears that operation

from the thigh is regarded as entirely satisfactory, though Masson weakens his opinion in its favour by advising that, after the method of Coley, the pectineus muscle and the wall of the crural canal should be brought together with a mattress stitch. We must confess some disappointment that the inguinal operation does not commend itself to the surgeons of the Clinic, as we hoped to glean from this article some clinical evidence as to its value. Giffin's papers on *Splenectomy* crystallize views founded on huge experience as to indications for and the value of this operation, and bring the immediate results of 245 splenectomies up to date. A few years ago it seemed from reports from the Mayo Clinic that splenectomy, when the spleen had been reduced by preliminary use of radium, held out some hope for patients suffering from myelogenous leukaemia. Giffin's opinion now seems to be that it is of doubtful value. The most satisfactory conclusion to be gathered from these articles is from a second report, which includes over 50 cases, in which the operation is shown to be of considerable value in pernicious anaemia. It is argued that, as in splenic anaemia, the indication for and value of splenectomy in pernicious anaemia is undue haemolysis—taking the bile pigments as an indication of hemolytic activity, evidence is deduced that at least a temporary reduction of the hemolytic factor occurs in a very large proportion of cases.

Les Occlusions Aigues et Subaigues de l'Intestin. By A. C. GUILLAUME. Pp. 304, with 21 illustrations. 1922. Paris: Masson et Cie. 12 fr. net.

THE author's object in this monograph has been to correlate the clinical and pathological features of acute and subacute intestinal occlusion with the results of experimental investigations, and to found thereon a rational therapeutics. As is usual in France the term 'occlusion' is limited to the acute or subacute condition, the chronic form being designated 'obstruction'. It is rightly pointed out that intestinal occlusion is not a pathological entity, but merely a syndrome—a complication which may supervene in the course of very diverse affections. Three groups of ileus are described: (1) Ileus by stimulation, where the bowel is occluded by approximation of its walls; (2) Ileus by obturation, where the bowel is blocked by a body independent of the wall and mobile within the lumen; and (3) Paralytic ileus. Of these the first and second are usually grouped together as 'organic' or 'mechanical' ileus, in contrast with the third, which is described as 'functional' or 'dynamic' ileus, that two or more of these factors may be, and in practice are usually found associated is properly insisted upon. We do not, however, consider that sufficient emphasis is laid upon the distinction between the earlier and local manifestations and the later general disturbances consequent upon them, and we prefer to limit the term 'acute ileus' to the latter, the local condition being referred to as 'acute intestinal obstruction'. Thus 'acute ileus' is the general condition brought about when a local 'acute intestinal obstruction' has been in existence for a sufficient length of time, and the whole trend of modern teaching is to prevent its supervention in any given case by means of early diagnosis and surgical treatment.

The work is divided into six chapters, dealing respectively with: (1) The general pathological anatomy of occlusion; (2) The clinical features; (3) Prognosis; (4) Diagnosis; (5) Physiology; and (6) Treatment. The general pathological features are well and concisely described, and attention is drawn to the changes occurring at a distance from the site of occlusion as for example, the frequency of gangrene of the caecum in obstructions of the sigmoid colon. The clinical signs associated with the various causes of occlusion are fully detailed, and are illustrated by typical case histories, with operative findings. Particularly worthy of mention are the descriptions of occlusion from gall stones and from lesions of the mesenteric vessels—embolism, thrombosis, and arteriosclerosis. It is shown that arteriosclerosis of the mesenteric vessels is much less uncommon than is usually supposed, and may occur in a localized form without any evidence of such changes in the radial or other artery accessible to palpation. Its importance in relation to embolism is duly stressed, and doubt is thrown upon the possibility of ileus from a single embolus in a mesenteric vessel unless it be the site of previous arteriosclerosis. Slight degrees of vascular ileus are met with comparable with the intermittent claudication of Charcot in the lower limbs, and arising from arterial spasm superimposed upon arteriosclerotic changes. Resection of the entire gangrenous area is the method of choice at present in the treatment of vascular ileus, although the mortality is given as 79 per cent. The question is discussed of possible operation upon the vessels themselves—the removal of the embolus, sympathectomy of the nerves of the vessels.

The chapter on prognosis is one of the best, and is based upon an analysis of over 700 cases of occlusion and 200 cases of strangulated hernia, the most recently published series of cases being included. The very much better prognosis of occlusion from external than from internal cause (such as strangulated hernia) is shown by a careful analysis of statistics to be due almost entirely to the difference in time before the condition is recognized and surgically treated, and a very strong plea is made for early diagnosis and early surgical intervention.

In discussing diagnosis, the value of auscultation of the abdomen—so often overlooked—is noted, and the importance of not waiting for the onset of fecal vomiting in any doubtful case is rightly insisted upon. Greater value than we are inclined to assign is given to radiography after opaque meal or enemata, as a diagnostic measure in acute and subacute ileus the examination being

wide in the recumbent position—this however is to be avoided if perforation of any part of the alimentary canal is suspected. It is urged that it is not more dangerous to move a patient to the radiological examination table than to the operation theatre.

In the section on physiopathology great stress is laid on the capital part played by toxic absorption from the contents of the bowel proximal to the obstruction and in my post-operative deaths are attributed to massive intoxication. The absorptive power of the bowel above the obstruction is much diminished whereas that below remains unimpaired, when therefore the obstruction is relieved and the contents of the distended bowel are allowed to escape into the bowel below toxins are rapidly absorbed and massive intoxication results. On this in the section on treatment is based a very strong plea for the examination of the distended bowel by a small trocar before searching for the cause of the obstruction and it is also advanced as a strong argument against enterenterostomy in the neighbourhood of the obstruction. Since paralytic ileus often persists after removal of the cause of the obstruction it is recommended to leave a temporary fecal fistula just above the site of the obstruction—in the large intestine by tying in a Paul's tube in the small intestine by burying a small catheter in the bowel wall after the method of Witzel in gastrostomy. Exploratory laparotomy is advised in all cases of functional ileus since in a series of thirty cases it was found not to increase the risks while it gives the security that in organic lesion is not being overlooked.

While this book does not introduce any strikingly new features yet we can confidently recommend it as a very up to date and lucid exposition of the whole question of intestinal occlusion.

Rickets: A Study of Economic Conditions and their Effects on the Health of the Nation

By J. LAWSON DICK, M.D. F.R.C.S. 8vo. Pp. 185. Two parts combined in one volume. Illustrated. 1922. London: Wm. Heinemann Ltd. 25s. net.

It was no disease that has attracted the attention of the clinician and the laboratory worker during recent years more than rickets. The reason is not far to seek. It is a disease that struggles against its vitality at its source and, as Sir William Osler once said, it is the greatest indirect cause of infant mortality in this country.

The monograph entitled *Rickets*, written by Mr. Lawson Dick, is a book of nearly 500 pages, and it would be difficult to find a single page that could be dispensed with. The time is ripe for such a book, and the author has successfully brought together information from near and far and presented the profession with a treatise that should prove of great value to the cause of preventive medicine and national hygiene. The subject has been approached with wide vision and from the broadest standpoint, and the author has spared himself no pains in collecting evidence about the disease from many diverse sources.

The first part of the monograph deals with the world distribution of the disease and with its signs and symptoms. It may be said at once, from a perusal of this part of the book, that one is inevitably forced to the conclusion that the disease is one confined mainly to those parts of the world where dense masses of population are congregated together in industrial centres where sunshine is lacking, overcrowding is rampant, and the winters are long and trying. One is not long in waking up to the fact that Mr. Lawson Dick is a whole-hearted supporter of the environmental factor of causation, and while many will be unprepared at the present time to accept this theory so wholeheartedly as the author, yet there can be little doubt that the evidence so ably presented by him throughout the book will convert many waverers to his side. The etiology of the disease is dealt with more especially in the second half of the book where the experimental evidence is examined, and a legitimate criticism would be that while the dietetic theory is fully examined and discussed, yet in the end this factor is too easily put aside as of quite secondary importance in the development of the disease. We agree with the author that the dietetic theory fails to account for a large amount of rickets, and that defective environment and bad hygienic conditions more readily fit the situation, but we do feel that there is a risk of allowing the pendulum to swing too far in the opposite direction. There are cases of rickets that one is unable to explain by the environmental factor of causation, and there are children showing no evidence of the disease whose environment is so bad that it would seem on this theory to have been impossible for them to escape. While the actual determining factor still remains unsolved it would seem wise to believe that both factors may be at work—the dietetic one becoming active when the environment and hygienic conditions are of the necessary type.

In the description of the signs and symptoms of rickets much valuable information is forthcoming. In discussing the earliest signs we are not sure that the author does not spend his net too wide, and it is doubtful whether all will agree with him when, on page 102, he writes that

the changes begin as a rule almost immediately after birth or within the last few weeks of extra-uterine life. There has recently been in some quarters a tendency to look upon most of the abnormalities of young infants as being of syphilitic origin, and it would be a pity if the same tendency were to show itself when dealing with rickets. Especially would this be so if the environment theory of causation were finally accepted. Physical and constitutional abnormalities certainly due to defect of diet might then run the risk of being included under the heading of rickets and the importance of the diet gradually lost sight of.

The chapter dealing with the teeth in rickets is most acceptable and is a part of the subject to which the author has given prolonged attention. From what he writes in the latter part of the book, where he deals with etiology, it would appear that he does not consider the changes in the teeth brought about by the experimental feeding of puppies during the investigations carried out by Dr. and Mrs. Mellin as identical with the hypoplastic changes of true rickets. We are not convinced that the results of these important experiments can be so easily put aside as having little bearing on true rickets of childhood.

In the second part of the book the history of the disease is dealt with in a most interesting way, and the chapter "Glisson and his Times" is one of the most readable in the monograph. The literature of the disease is very fully given and will be of considerable value to future investigators.

The sections of the work which are of more exclusively surgical interest are those dealing with the mechanics of the development of deformities and with their subsequent treatment. It is easy to understand how bow leg or knock knee may progress, once a bias in the given direction has been acquired. The determination of this bias is convincingly explained by showing that the infant who shuffles will become bow legged, while the infant who crawls will become knock kneed. In considering deflection at the knee after walking has begun, one may, however, be permitted to doubt whether habitual eversion of the feet will tend to bow legs while returning the feet parallel will incline to knock knee deformity. The association of knock knee with flat foot is a well established fact, and one may more readily believe that the 'position of rest' with everted feet will increase the strain on the internal lateral ligaments of the knees and lead to decreased pressure on the inner condyle, with its resulting overgrowth and knock knee. The author definitely maintains the view that the static deformities of adolescents are mainly rickets in origin, either through the disease beginning at about puberty through a recrudescence of early disease, or through weakness of muscles, ligaments or bones as a legacy of rickets in earlier life.

In a general work on rickets such as this, it is perhaps well that the author should avoid giving a full text book account of surgical treatment such as is the province of a work on orthopedics. This aspect of the disease is dismissed in a few pages in which surgical principles are enunciated in a sound manner, but without sufficient detail to make them of more than suggestive value.

In conclusion, we would go so far as to say that we look upon this book as a milestone in the history and literature of this disease. It certainly will take its place as an important contribution to the cause of preventive medicine.

Text-book of Surgery for Students and Practitioners. By JOHN A. C. MACLEOD, M.B., C.M., Senior Assistant to the Regius Professor of Surgery in the University of Glasgow. 8vo. Pp. 619 + xvi, with 333 illustrations. 1922. Glasgow: Maclehose, Jackson & Co. 30s. net.

THE volume before us adds one more to the many text books of general surgery for students and practitioners, it has been produced at the request of many of the author's pupils and is founded on the notes used by him in teaching. Within its six hundred pages is contained a very concise statement of general surgical affections and principles, affections of the various tissues, and regional surgery. Fractures and dislocations are omitted, as the author has already dealt with these in a separate manual. The general impression left by a study of the text book is that on the whole it is good but that the author has been carried away by his desire to omit nothing and has thus been led to include many more conditions of little practical importance to a student and to give such short accounts of various methods of treatment that they are of little use to the practitioner. In this respect the author might wisely have consulted his own judgement rather than, as he states in his preface, have considered the requirements of examiners, particularly those who have done little or no teaching. Surely this variety of examiner must be a very rare specimen and hardly worthy of consideration! It thus comes about that in many places the reader finds a straggling amount of proportion, as, for instance, when twice as many lines are devoted to dislocation of the penis as to circumcision of that organ.

In view of the vastness of the subject and the limitations of space, short dogmatic statements are unavoidable, and, speaking generally, these statements in most instances reflect the accepted teaching. On the other hand, there are many statements in the book to which the majority of surgeons would probably take exception. Thus in the treatment of senile gangrene, the author states that it is rarely wise to amputate when the gangrene is spreading, as gangrene is apt to recur in the stump, that when amputation is performed it should generally be done at the knee, and that the flaps should not be sutured, but extended gently by strapping. Most surgeons it is believed, would advise amputation in the majority of cases when the gangrene is spreading beyond the toes, would remove the limb through the lower part of the thigh and would run it primary union of the wound by accurate suturing of the flaps. In connection with tuberculous disease of the spine the student would certainly conclude that the most common cause of pressure paraplegia is acute angulation, although occasionally pus from erosion of the vertebra may work backwards, etc. In dealing with syphilitic affections of joints, painless effusion, especially in

the knee and sometimes bilateral is mentioned as a late secondary manifestation of the acquired disease. It would have been more accurate to describe this form under the inherited type and to refer to its almost constant association with interstitial keratitis. Again speaking of a tendon sheath is it quite certain that a simple ganglion is a protrusion of synovial membrane through an aperture in its fibrous envelope?

In some practical advice on the examination of the breast, especially in the presence of a tumour, the importance of the recumbent position is not mentioned and indeed, as it is stated that in examining the axilla the arm should be hanging by the side it may be assumed that the patient is seated. It would be interesting to know whether the author has really seen relief of threatened suffocation due to cancer en masse by making long incisions through the skin of the chest wall. We would take serious exception to the inclusion of acute appendicitis with strangulated hernia as the two most common causes of acute intestinal obstruction. In the practical consideration of an acute abdominal case most of the common conditions belong to one or other of two great classes, one in which a mechanical obstruction is present and the other in which some form of acute peritoneal infection has occurred. Strangulated hernia belongs to one class and acute appendicitis to the other. The above are a few only of the statements to which exception may be taken, but even if all were mentioned there would still remain a large majority with which we entirely agree.

Finally, a few words may be said of the illustrations, which number five hundred and thirty-five. The best substitute for a real patient or specimen is a good illustration, and of these before us it will be agreed that the majority, although small, are really useful and their collection must have been no small labour to the author. On one page is a very useful group of four figures illustrating conditions which may resemble a strangulated hernia. We cannot however avoid the conclusion that many of the figures are useless. Acute inflammatory conditions rarely lend themselves to satisfactory illustration and it may be doubted whether such figures as those of acute pyogenic ischio-rectal abscess and large carbuncle of the neck are of any value. The same applies to some of the reproductions of radiograms and microscopic sections and in many instances it requires a keen sense of imagination to discover the points which the illustration is intended to show.

The conclusion is that in a later edition of this text book improvement might best be effected by a judicious removal of matter which is practically unimportant and a more ample consideration of what remains.

Artificial Limbs and Amputation Stumps. A Practical Handbook. By L. MURRAY LITTLE, F.R.C.S. etc. Demy 8vo. Pp. 319. 5s. with 267 illustrations. 1922. London: H. K. Lewis & Co. Ltd.

MR. MURRAY LITTLE has had a unique experience in dealing with the fitting of various types of prostheses during the last seven years, and the book is the outcome of his labours. He modestly states that he desires to record the conclusions that he amongst others has reached in the treatment of amputation stumps, and in the prescription and supervision of prostheses of some 25,000 cases. He does not pretend to offer a complete encyclopaedic work, such as has been recently produced in Germany, but has restricted himself largely to British practice as laid down by the Ministry of Pensions, and he has also endeavoured to avert the danger of the book becoming merely a compilation of limb makers' catalogues. In both considerations he is to be congratulated, as the reader will wish to acquaint himself only with the most successful models and procedures, and surgeons will desire to fashion their work to the best advantage from the limb makers' standpoint.

The historical chapter is a delight, and the illustrations and description of artificial limbs of the time of Ambrose Pare bear striking resemblance to certain present-day types.

When dealing with amputation stumps one wishes that the author had laid down a few general principles for the avoidance of joint contractures, instead of merely mentioning the existence of such preventable deformities. He omits to mention that it has sometimes been necessary to divide the posterior ligament of the knee joint in contracture following below knee amputations where conservative methods of stretching have failed.

Mr. Little is a great advocate of end bearing stumps in the leg and thigh, but however excellent in theory weight bearing 'pad and sling' may be, the experiences of other surgeons do not quite coincide with the statistics given on page 50.

The chapter on cinematization makes sad reading, as the purely prosthetic difficulties have not been overcome. Despite the fact that work is still in progress in the experimental department of the Pensions Ministry, it is probable that the procedure will sink into oblivion.

A large amount of space and many illustrations are devoted to arm prostheses, the modern trend being towards working arms, heavy or light, with simple attachments, or to light dress arms of 'certalind', which can be worn at the end of the day's work, or entirely for sedentary occupations.

As regards artificial hands, the author says truly that they serve to some extent to mask the mutilation, and are occasionally useful. It is a matter of regret that more than this cannot

be said conscientiously of this part of the prosthesis, despite the ingenuity and industry of engineers and limb makers during five centuries and especially during the last five years."

An interesting section on the relationship of normal gait and gait with an artificial leg contains the observations of du Bois Reymond, and the later experiments of the Munitions Inventions Department which were conducted with the aid of a slow moving cinematograph.

Mr Little has always been an advocate of a light limb, and the trend of modern design has had this aim in view. Prosthetic designs improve so rapidly that the book is already in danger of being out of date in this respect. A modification of the light duralumin Desoutter Limb with a wooden bucket is now being supplied in large numbers to pensioners. Other makers are bringing out similar types, so that the appendix devoted to specifications of the Standard Government Limb will before long require revision.

Mr Little pays a graceful tribute to the progressive spirit displayed by the Pensions Ministry in its prosthetic policy. His innate modesty forbids him to state that he himself helped to mould it.

The book will rank as a standard work on a subject which requires for authorship a deep mechanical knowledge.

Surgical Pathology [Students Synopsis Series] By ERIC PLARCE GOULD, M.D. F.R.C.S.
Crown 8vo Pp 160+vi 1922 London J and A Churchill 6s net

THIS is a little book of 165 pages which can be bought for six shillings. It belongs to the Students Synopsis Series and conforms to type.

The fact that such books continue to be printed and are bought by successive generations of students proves that there is a demand for them, and they seem to fill a gap which exists either in the teaching given or in the minds of the students. Of its kind the book is quite good and one can only hope that it is the prelude to some more enduring work from its author.



PERCIVALL POTT

1714 1788

*Copied in permission from the painting by George Romney in the Council Room
of the Royal College of Surgeons of England*

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EPONYMS

By SIR D'ARCY POWELL, K.B.L., LONDON

VII PERCIVALL POTT· HIS OWN FRACTURE

PERCIVALL POTT ranks high amongst the surgeons of the eighteenth century. He was a sound teacher of clinical surgery when the organized teaching of medical students had hardly yet begun. Samuel Sharp, of Guy's, was before him in point of time, but Sharp taught those who were already in practice. "He gave," says the historian, "a course of anatomical lectures to which were added the operations of surgery with the application of bandages, to a Society of Naval Surgeons which met in Covent Garden on winter evenings." This course he repeated for several years, and when attacks of asthma caused him to discontinue it, the school was carried on by William Hunter, who taught surgery as a branch of anatomy.

Edward Nourse, to whom Pott was apprenticed, gave occasional lectures to the students at St. Bartholomew's Hospital, but they dealt rather with the principles than the clinical aspects of surgery, and were of a purely formal character if we may judge from the syllabus of his lectures on anatomy which is still extant.

Pott took the modern line. Attached to a large hospital where there was plenty of clinical material, he used his opportunities by telling students of the mistakes he had made, of the cures he had done, and of the cases he had seen. He was necessarily ignorant of surgical pathology, for it was born of John Hunter, who attended some of these lectures as his pupil. It is curious to observe in reading Pott's treatises how largely this ignorance vitiated his conclusions and paralyzed his treatment. We must think of him, therefore, as a surgeon of the old school, as superior to Wiseman as Wiseman was to Woodall, but in every respect inferior to John Hunter, who was a thinker as well as an observer. Nevertheless Pott's sound common sense, his transparent honesty of purpose, his desire to teach what he knew, and his position as surgeon to a large hospital, made him a great leader in surgery whilst the success of his lectures and his pleasant manner of writing spread his teaching widely and made his name known throughout France and Germany.

Percivall Pott was born in London, the son of a scrivener who died when he was three years old, leaving his mother so poor that after his death a small box was found which contained less than five pounds, the whole sum received from the wreck of his father's fortune. His mother, however, was well connected, her first husband had been a Houblon, and the Houblons were merchant strangers under Elizabeth, Roundheads under the Commonwealth, Whigs and founders of the Bank of England under William III. His upbringing was easy, therefore, and in 1729 he was bound apprentice to Edward Nourse, then Assistant Surgeon to St. Bartholomew's Hospital, paying two hundred

guineas for his indentures. He appears to have acquired a reputation unusually early, for during the later years of his apprenticeship it is recorded that, "being confident in the fair prospect of industry, he hired a house of considerable size in Fenchurch Street and took with him his mother" and his step sister. The venture was successful, for at the end of his apprenticeship in 1736 he was made free of the Barber Surgeons' Company, and there is an unusual entry in the minute books of the Company about him. It states that "At a meeting of the Court of Examiners on September 7, 1736, the Question being put whether Mr Percival Pott should be examined at this Court he not having waited on all the Governors and Examiners to desire the favour of their presence at his examination, and it appearing to the Court that Mr Pott had been sent for out of Town to attend Sir Robert Goodesall's* Lady where he was detained so long as not to be able to return within the time limited for his attendance on the Governors and Examiners, and Mr warden Petty having been pleased to say that he would make his excuse to the Court, It was resolved that the Court would proceed to the Examination of the said Mr Pott notwithstanding his default in attending the Examiners, but this is not to be a precedent in time to come to any other person,—And then

"The said Mr Percival Pott was examined touching his skill in surgery in order to have the Great Diploma, his answers were approved, and he was ordered a Diploma under the seal of the Company and the hands of the Governors testifying his skill and Impowering him to practise"

This minute is interesting from many points of view. It shows that he had already determined to practise pure surgery, as the Great Diploma corresponded in some measure to the present F R C S. It was rarely given, and only after a very thorough examination. It raises the suspicion, too, that he was practising midwifery at this time, which he would be entitled to do if he chose, as neither the College of Physicians, the Company of Barber Surgeons, nor the Universities of Oxford or Cambridge, claimed any control over a man midwife. It shows, too, that Pott was already esteemed by the Barber Surgeons' Company, for so old and well-established a custom as the personal visit to the examiners would not otherwise have been waived.

Pott was a devoted son, and so long as his mother lived he made a home for her and it was not until after her death that he married Sarah Cruttenden, by whom he had five sons and four daughters. He then moved into a house in Watling Street, where he began the course of surgical lectures which made him famous. He was elected Assistant Surgeon to St Bartholomew's Hospital in 1744, becoming full surgeon in 1749.

In 1756 an accident befell him which—by a curious confusion of thought—made his name of world-wide fame. 'As he was riding in Kent-street, Southwark', says Sir James Earle, his son-in-law, successor, and biographer, "he was thrown from his horse, and suffered a compound fracture of the leg, the bone being forced through the integuments. Conscious of the dangers attendant on fractures of this nature, and thoroughly aware how much they may be increased by rough treatment, or improper position, he would not suffer himself to be moved until he had made the necessary dispositions. He sent to Westminster, then the nearest place, for two chairmen, to bring their poles, and patiently lay on the cold pavement, it being the middle of January, till they arrived. In this situation he purchased a door, to which he made them nail their poles. When all was ready, he caused himself to be laid on it, and was carried through Southwark, over London-bridge to Watling-Street, near St Paul's, where he had lived for some time—a tremendous distance in such a state! I cannot forbear remarking, that on such occasions a coach is too frequently employed, the jolting motion of which, with the unavoidable awkwardness of position and the difficulty of getting in and out, cause a great and often a fatal aggravation of the mischief. At a consultation of surgeons, the case was thought

* Robert Goodschall Alderman of Bishopsgate Ward and Sheriff of London received the honour of Knighthood at St James's Palace on Oct 31 1735 on the occasion of an address congratulating King George II on his safe return from Hanover. Goodschall was afterwards M.P. for the City of London. He died in 1742 whilst holding the office of Lord Mayor. Lady Goodschall died Sept 27, 1750.

so desperate as to require immediate amputation. Mr Pott, convinced that no one could be a proper judge in his own case submitted to their opinion, and the instruments were actually got ready when Mr Nourse who had been prevented from coming sooner fortunately entered the room. After examining the limb he conceived there was a possibility of preserving it. In attempt to save it was acquiesced in, and succeeded. This case, which Mr Pott sometimes referred to, was a strong instance of the great advantage of preventing the insinuation of air into the wound of a compound fracture and it probably would not have ended so happily, if the bone had not made its exit, or external opening, at a distance from the fracture, so that when it was returned into the proper place, a sort of valve was formed, which excluded air. Thus no bad symptom ensued, but the wound healed, in some measure by the first intention.—The appearance of Mr Pott as an author was an immediate effect of this accident. It is clear from this account that the accident which Pott sustained was an open fracture of the tibia—spiral or very oblique—and that the knob shaped end of the upper fragment penetrated the skin.

Pott's fracture, as it is now known he described carefully in his *Remarks upon Fractures and Dislocations*, published in 1768 without any reference to his own case. The desperate nature of the prognosis in cases of compound fracture as it was then treated is well exemplified by Pott in this treatise. He wrote "When a surgeon says that a limb, which has just suffered a particular kind of compound fracture ought rather to be immediately cut off than that any attempt should be made for its preservation he does not mean, by so saying, that it is absolutely impossible for such limb to be preserved, at all events he is not to be supposed to mean so much in general, though sometimes even that will be obvious, all that he can truly and justly mean is that from the experience of all time it has been found, that the attempts to preserve limbs so circumstanced, have most frequently been frustrated by the death of the patients, in consequence of such injury, and that from the same experience it has been found, that the chance of death from amputation is by no means equal to that arising from such kind of fracture."

This passage gives a good example both of Pott's literary style and of the sound common sense with which he was endowed. He resigned his office of Surgeon to St Bartholomew's Hospital in 1787, after having served the charity, as he used to say, man and boy for half a century. He died of pneumonia on December 22, 1788, and was buried in the chancel of St Mary's, Aldermary, in Queen Victoria Street, where a tablet to his memory may still be seen on the wall of the south aisle. His kindness of heart was proverbial, and although he had a large family dependent upon him, it is said that, at one time, he had three needy surgeons living in his house until he could provide them with the means of earning an independent livelihood. His high character and blameless life helped to raise the surgeon's social standard in this country.

The portrait is copied by permission from that in the Council Room of the Royal College of Surgeons of England. It was painted by George Romney, and was presented to the College by the Ven Archdeacon J H Pott. The College also possesses a life size half-length portrait painted by Sir Nathaniel Dance Holland, Bt, R A, and there is the well known picture by Sir Joshua Reynolds, which hangs in the Great Hall at St Bartholomew's Hospital.

ABNORMALITIES OF THE DUODENUM

By JOHN H. ANDERSON, C.M.G., C.B.E., MELBOURNE

ARISING from certain remarks made by a Melbourne clinician, a routine examination was made of 100 consecutive subjects presenting in the Anatomy Department of the University of Melbourne, with a view to noting any cases of gross abnormality in the shape or position of the duodenum. As a result of this examination four cases of gross abnormality were discovered, which may be classified as follows —

Case 1 — Abnormal shape

, 2 — Constriction caused by the superior mesenteric artery

„ 3 — „ „ „ „ an annular pancreas

„ 4 — Abnormal position

General Consideration of the Cases Presenting Abnormality —

Preservative — All cases had been formalin hardened

Cause of Death — This varied from cardiac failure to senile decay, but in no case had it any apparent connection with the abnormality present

Sex Incidence — All cases occurred in males

Age Incidence — All cases were in old people, the ages ranging from 65 to 74 years

Clinical History — The sources from which the material for a dissecting-room is gathered make the collection of medical histories a matter of some difficulty. From what was available, however, the interesting fact emerged that the duodenal abnormalities had not given rise to any acute trouble during the later years of life. The age of the subjects and the absence of signs of operative interference would tend to support this observation.

The Normal Duodenum — The description of the duodenum as set out in Berry's *Practical Anatomy*¹ and Gray's *Anatomy*² has been taken as a criterion for comparison and is regarded as presenting the normal state of affairs.

PARTICULARS OF CASES

Case 1 — Abnormal Shape

GENERAL DESCRIPTION — The duodenum consisted of four parts, superior, descending, ascending, and horizontal, in that order. The superior or first part was normal, and passed directly into a second or descending part, which after a caudal course of 6 cm terminated in an acute bend cranially and to the right, with a slight dorsal inclination, so that the left margin of the ascending part was overlapped by the right margin of the adjacent descending part. The ascending part, after a course of 5 cm, turned to the left at an angle of 90° and passed dorsal to the descending part, thus forming the commencement of the fourth or horizontal part, which after a straight course of 12 cm terminated in the usual manner, though slightly more cranial than normal. The diameter of the bowel was within normal limits throughout.

RELATIONS — The ascending part lay ventral to the caudal pole of the right kidney, and was in close contact with the hilus of the same viscus. The pancreas lay cranial to the horizontal part, and though no head could be defined, an uncinate process was distinctly visible. The superior mesenteric vessels were normal, and passed ventral to the horizontal part of the bowel. The common bile duct and the main duct of the pancreas united in the substance of the latter viscus, and the resulting single duct passed ventral

to the horizontal part of the duodenum to open into the lumen of the bowel on the dorsal aspect of the acute flexure caused by the union of the descending and ascending parts of the duodenum. These points are well seen in the diagram attached (Fig 254), which is about one quarter life size and was constructed from actual photographs.

REMARKS—Piersol³ states, "much variation exists in the shape of the duodenum, and thinks this may be due "to an unusually long duodenum, which, after having completed the usual course, describes one or more additional curves before reaching the duodenojejunal flexure. Such would not appear to be the cause in this case where the whole duodenum presented a normal length of 25 cm, and where the operating curve would appear to be a kink taking place during rotation. A careful search of available literature has failed to reveal an absolutely similar case, though that described by Schaeffer-Decker,⁴ and quoted and illustrated by Piersol,³ is a near approach. The position of the conjoined duct formed by the union of the common bile-duct and the main duct of the pancreas is a point of some clinical importance.

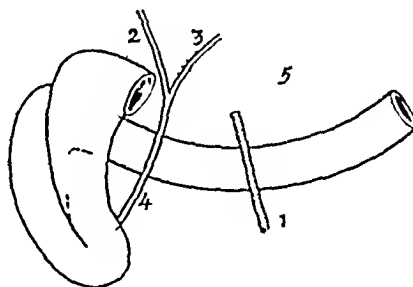


Fig 254—Case 1 (1) Superior mesenteric artery (2) Common bile duct (3) Main pancreatic duct (4) Combined bile and pancreatic duct (5) Pylorus

Case 2—Constriction caused by Superior Mesenteric Artery

GENERAL DESCRIPTION—In five cases of the hundred examined there was some 'flattening' observed at the point where the superior mesenteric vessels crossed the horizontal part of the duodenum, and in one other case a definite constriction was noticed at the same point. In this last case the duodenum was normal elsewhere as regards length and diameter, and there was no 'chronic dilatation of the first three parts' as described by Wilkie.⁵ The constriction was an annular one, the outside diameter being 0.9 cm. On opening the bowel it was found that a slate pencil would pass freely through the lumen of the constriction, but a lead pencil required some force to push it through, and caused an increase in the outside diameter. The peritoneum over the constriction was particularly smooth and shiny, and "looked worn", and there was an absence of any puckering. Macroscopic examination of the constriction, after longitudinal section, failed to show any abnormality in the bowel wall, nor could any trace of ulcer be found in stomach or duodenum. The stomach was not dilated, and the superior mesenteric artery, in common with other abdominal contents, appeared normal.

REMARKS—The interest of this case lies in its presence in a male, the absence of gastric or duodenal ulcer, the absence of gastric or duodenal dilatation, and, as far as can be ascertained, the absence of clinical disturbance.

Bearing in mind the danger of drawing conclusions from a single example (and that a formalin-hardened dissecting room subject), it would appear from the cases put forward by Wilkie⁵ and Devine⁶ that three grades of this type may be described—

Grade I—Constriction, without duodenal dilatation and without clinical signs.

Grade II—Constriction, with moderate duodenal dilatation and perhaps ulcer (gastric or duodenal), and with clinical signs of a chronic nature.

Grade III—Constriction, with excessive duodenal dilatation, and with clinical signs of an acute nature, mainly those of obstruction.

Case 3—Constriction caused by Annular Pancreas (Figs 255, 256, 257, 258)

GENERAL DESCRIPTION—A complete ring of pancreatic tissue surrounded the descending part of the duodenum, causing a very definite constriction. The greatest diameter of this ring, which was flattened in the ventrodorsal direction, was 2.5 cm and the smallest diameter was 1.8 cm. The rest of the pancreas was normal. The first part of the duodenum could not be defined as such, but between the pylorus and the duodenal

constriction was a dilated portion of bowel 4.7 cm long and 4.8 cm in diameter. The constriction itself had an outside diameter of 1.2 cm and would admit a lead pencil through its lumen. Below the constriction the duodenum had a diameter of 3.5 cm, and the total length of first and second parts combined was 10.5 cm. Opposite the crossing point of the mesenteric vessels the diameter fell to 2.6 cm, swelling out to 3.5 cm just proximal to the duodenojejunal flexure. The total length of the whole duodenum was 25.5 cm.

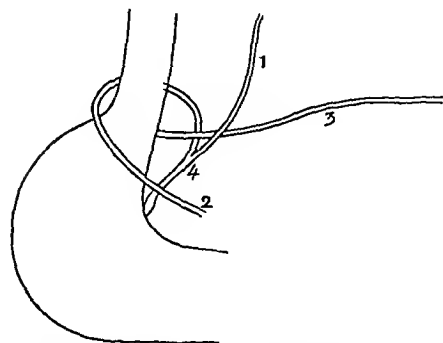


Fig. 250.—Case 3. (1) Common bile duct (2) Main pancreatic duct annular in form (3) Large accessory pancreatic duct (4) Junction of bile and pancreatic ducts

Arrangement of Ducts—The main pancreatic duct started in the caudal part of the head of the pancreas, and passed to the right, completely encircling the duodenum in the ring of pancreatic tissue. It then joined the common bile duct, and opened into the bowel below the constriction and on its medial side. The remainder of the pancreas was drained by an accessory duct, very well developed which opened into the bowel below the constriction and 2 cm cranial to the opening of the conjoint duct above described. This arrangement of the ducts is shown in Fig. 255 (not drawn to scale).

REMARKS—Annular pancreas is sufficiently rare to deserve some special consideration. A

careful search of the available literature has revealed fourteen cases, some of which are summarized in the following table.

CASES OF ANNULAR PANCREAS

AUTHOR	SEX	PART OF DUODENUM	STATE AT RING	STATE ABOVE RING	STATE OF STOMACH	DUCT IN RING
Ecker ⁷	M	Second	Constricted	Dilated	—	Opens into main duct
Auberg ⁸	M	Fourth	Constricted	—	—	—
Symington ⁹	M	Second	Constricted	Dilated	Normal	—
Genersich ⁸	M	Second	Constricted	Dilated	Dilated	Opens into main duct
Sandras ⁸	M	Second	Constricted	Dilated	—	—
Tiel en	M	Second	Constricted	Dilated	Dilated and hypertrophied walls	Normal
Baldwin ⁸	—	Second	Constricted	Dilated	Normal	Opens into main duct
Cords ¹¹	M	Second	Constricted	—	—	One opening main duct one opening separately
Anderson	M	Second	Constricted	Dilated	Dilated	Forms the main duct

Cases of annular pancreas are also reported by the following, but full particulars are not available in Melbourne. Sumner,⁸ Thacher,⁸ Lecco¹⁰ (two cases), Becourt,¹⁰ and Moyses¹⁵ Piersol,³ Quinn,¹² Cunningham,¹³ Poirier and Charpy,⁷ and Morris,¹⁴ among the text-books of anatomy, also refer to this 'most interesting but rare variation'.

Mode of Causation—This matter is fully discussed by Baldwin,⁸ Lecco¹⁶ and Cords¹¹. Baldwin's explanation (which seems fully borne out by the arrangement of the ducts) may be shortly stated as follows. The pancreas arises from the duodenal wall by two *Anlagen*, one ventral and one dorsal. The former consists of two parts, a left and a right

The right half is curved round to the right and then dorsal to the duodenum during the rotation of the latter and after fusion with the dorsal *Anlage* forms the caudal segment of the head of the pancreas. In it is developed the main pancreatic duct. The left half of the ventral *Anlage* generally atrophies. If it persists and extends to the left ventral to the duodenum, to join the main mass of pancreatic tissue or if there is an excessive growth of the right half of the same *Anlage* in a like direction, mural pancreas results.



Fig. 256—Case 3. Pancreas and duodenum, dorsal view. (A) Pancreas (C) Pancreatic ring, dorsal limb (D) Duodenum (E) Common bile-duct.

Summary—Annular pancreas is almost always found in males, and causes (a) A constriction of the second part of the duodenum, (b) A dilatation of the duodenum cranial to the constriction. This dilatation may involve the stomach and may be accompanied by hypertrophy of the walls of that viscus. There is generally a well marked duct in the ring of pancreatic tissue which

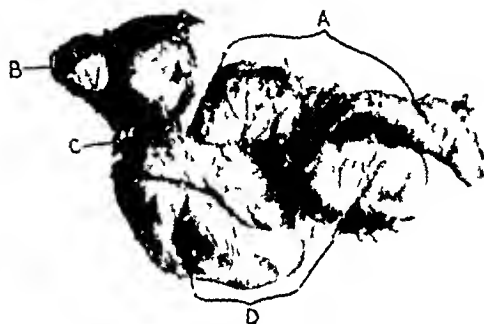


Fig. 257—Case 3. Pancreas and duodenum, ventral view. (A) Jejunum (B) Pylorus (C) Pancreatic ring, ventral limb (D) Duodenum.

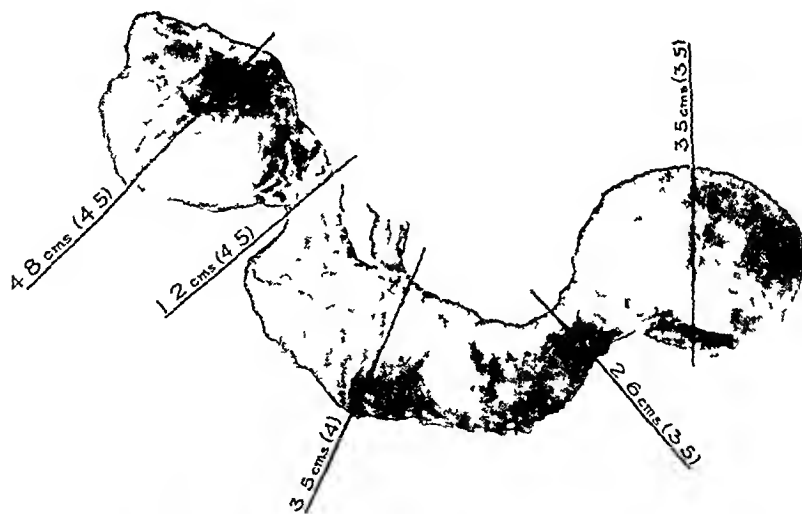


Fig. 258—Case 3. Duodenum after removal of annular pancreas. To show diameter of duodenum at various points. Normal diameters shown in brackets.

joins the common bile-duct. The mode of causation of this abnormality can be readily explained on embryological grounds.

CLINICAL CONSIDERATIONS—The surgical and anatomical literature available makes no mention of a clinical picture associated with annular pancreas, and this view is

supported by Huet,¹⁷ who states "Le pancreas annulaire peut rester longtemps sans symptômes chez le vivant, il peut être reconnu à l'occasion d'une intervention pratiquée par un syndrome de retrecissement pylorique ou de pancreatite chronique" This is in great contrast to the vivid picture painted by Wilkie¹⁸ of the possible effects of a constriction caused by the superior mesenteric vessels. The difference may lie in the comparative rarity of annular pancreas, and may be influenced by the fact that it produces a fixed constriction, not one liable to changes in calibre brought about by other abdominal variations, as would seem to be the case with a mesenteric constriction.

Case 4—Abnormal Position

GENERAL DESCRIPTION—An inguinal hernia, of the type described by Hamilton Russell¹⁸ as 'hernia magna', was present on the left side. This contained the terminal 15 cm. of the ileum, the cæcum and appendix, part of the ascending colon, and 30 cm. of the pelvic colon. With the exception of the pelvic colon, all the bowel within the sac

was attached to the peritoneum forming the dorsal wall of the sac by two well defined mesenteries, one for small and one for large intestine. The mesentery for the ileum appeared normal both within and without the sac, but between the two layers of mesentery of the large bowel there was deposited a large amount of fat. This mesentery ceased on passing from hernial sac to abdomen proper. The right colic flexure did not exist as such, but in the right iliac fossa the bowel made a gentle curve cranially and to the left, thus marking the commencement of the transverse colon. The left colic flexure was situated 25 cm. caudal to the lower pole of the left kidney. The pylorus lay opposite the third lumbar vertebra, and the duodenum crossed the mid line ventral and slightly caudal to the bifurcation of the abdominal aorta, at the level of the intervertebral disc between the fourth and fifth lumbar vertebrae. Fig. 259 shows roughly the position of the structures named. The liver, kidneys, and spleen were normal in position.

Though the number of lumbar vertebrae was normal, the distance between the tip of the xiphoid process and symphysis pubis was 8 cm. less than that observed on four other subjects of similar stature, while the tip of the last rib was almost in contact with the iliac crest on either side. The celiac artery was 5.5 cm. in length, and was directed caudally, ventral to an abnormally broad pancreas.

REMARKS—The peritoneal attachments of bowel to the dorsal abdominal wall would appear to have slipped, or else developed in an abnormal position, with a consequent descent of the various parts of the alimentary canal within the abdomen, up to but not including the left colic flexure. This in turn would cause the abnormal length of the celiac artery.

The suggestion is also advanced that the transference of so much bowel from the abdominal cavity to the hernial sac resulted in a lack of stimulus to longitudinal growth, which caused a reduction in the length of the long axis of the abdomen. If this supposition is correct, it has a certain bearing on operating for hernia of this size before body growth has ceased.

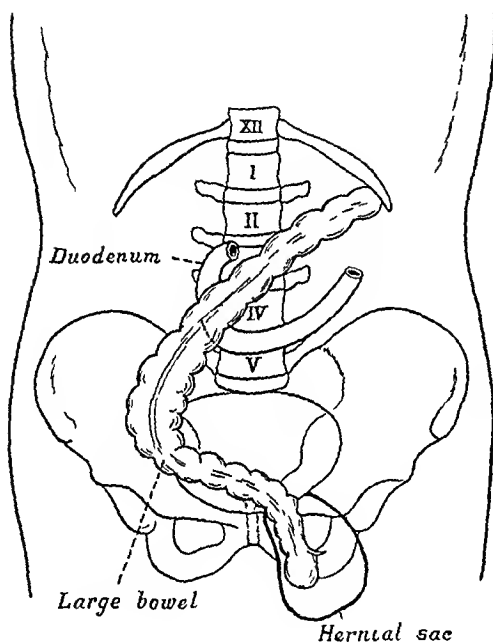


Fig. 259—Case 4

GENERAL CONCLUSIONS

In addition to the conclusions drawn with respect to the various cases described, it may be stated that —

1 Gross anatomical abnormalities of the duodenum are more frequent than is generally supposed

2 Such abnormalities may exist without producing any clinical evidence of their presence

I am indebted to Mr J C Eccles Professor in Anatomy, for his assistance in the dissecting carried out in these cases, and to Mr W H Preston for his admirable photographs

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LARGE MYELOID SARCOMA (MYELOMA) OF THE RADIUS IN WHICH THE TUMOUR IS WHITE THROUGHOUT.

By MATTHEW J STEWART, LEEDS

THE maroon colour of myeloid sarcoma is generally, and justifiably, held to be one of the most characteristic naked-eye features of these growths, and most surgeons would probably regard such an appearance at the time of operation as diagnostic. The dark-red or maroon colour may affect the whole tumour, or only a part of it, and it is quite usual to see considerable areas of white tissue here and there. As a rule, the latter correspond to the more fibrous portions in which giant cells are comparatively scanty, while the red parts are either very cellular areas, highly vascular, and with numerous multinucleated giant cells and many effused red blood corpuscles, or else mere hæmorrhagic extravasations of large size. I have long been familiar, however, with the fact that some of the white portions of a myeloid sarcoma, notably those occurring at the growing margin, consist, not of densely fibrous, comparatively acellular tissue, but of highly cellular, actively proliferating myeloid tissue, with a large proportion of giant cells. It therefore seemed reasonable to regard the maroon colour as a secondary, even accidental characteristic, due partly to increased vascularity and partly—and more especially—to extravasation of blood.

Under these circumstances, the finding of a large myeloid sarcoma which was white throughout was quite in keeping with one's preconception of the pathology of this tumour, but the rarity of the condition, as well as its theoretical importance, calls, I think, for a full and adequately illustrated case report.

While practically all the modern descriptions of myeloid sarcoma insist on the constancy of this colour characteristic, and only admit at most that *portions* of the tumour may be white, Sir James Paget,¹ in 1853, states quite unequivocally that 'the tumour may be all pale'. His description of the naked-eye characters of the growth is well worth quoting: "On section, the cut surfaces appear smooth, uniform, compact, shining, succulent, with a yellowish, not a creamy, fluid. A peculiar appearance is commonly given to these tumours by the cut surface presenting blotches of dark or livid crimson, or of a brownish or a brighter blood colour, or of a pale pink or of all these tints mingled, on the greyish-white or greenish colour basis. This is the character by which, I think, they may best be recognized with the naked eye, though there are diversities in the extent, and even in the existence, of the blotching. The tumour may be all pale, or have only a few points of ruddy blotching, or the cut surface may be nearly all suffused, or even the whole substance may have a dull Modena or crimson tinge, like the ruddy colour of a heart or that of the parenchyma of a spleen."

The case here recorded is a striking example of a myeloid sarcoma which is 'all pale'.

HISTORY OF CASE

The patient, a small girl of 6 years, was admitted to the Leeds General Infirmary under the care of Mr L. R. Brathwaite in March, 1922, suffering with a swelling of the distal half of the left forearm. This had commenced about three years before, and while it had grown slowly at first, during the last three months there had been a rapid increase in size. There was no history of injury, and there seemed to be little or no pain or discomfort. The tumour appeared to spring from the radius. It was firm, of regular, ovoid outline, and not tender on pressure. Several firm, enlarged glands were palpable in the left axilla.

An x-ray plate (Fig. 260) shows that the distal half of the diaphysis of the radius is the seat of radiolucent expansion. There is a very delicate shell of expanded bone round considerable portions of the periphery, with a number of slight trabecular thickenings on its inner aspect. On the side towards the ulna, however, the bony capsule appears to have completely disappeared.

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The central part of the tumour contains no bone whatever. Proximally, there is a fairly sharp line of demarcation between the tumour and the rest of the shaft, with a certain amount of bony superficial thickening under the periosteum, just where the shaft is beginning to be expanded distally. The tumour stops short at the limit of the epiphyseal cartilage. The appearances are very strongly suggestive, if not actually pathognomonic, of myeloid sarcoma.

On March 21 a small portion of the tumour was excised for microscopic examination. It was white in colour, and not at all like the usual appearance of a myeloid sarcoma, yet the microscopic characters were quite unmistakable (Fig. 262). In particular, osteoclast-like giant cells were found in the greatest profusion, constituting, in some fields, more than half the total area. At the point where the portion of

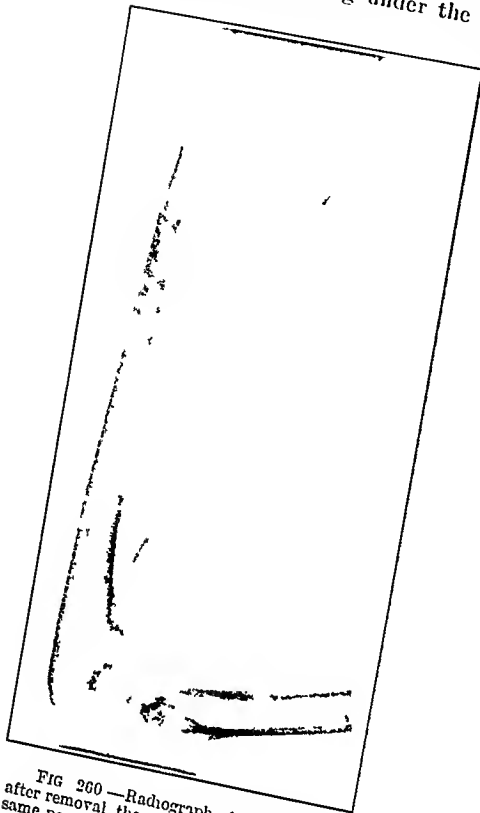


FIG. 260—Radiograph of the specimen after removal of the arm being roughly in the same position as in Fig. 261. The tumour is endosteal and radiolucent and it has caused great expansion of the shaft of the bone. A delicate shell of bone with trabecular markings is seen on the upper (radial) aspect of the growth, but on the side towards the ulna this capsule has completely disappeared.

growth was excised, there was no bony capsule whatever, the tumour was directly invading the surrounding muscle and other soft parts.

After careful consideration, it was decided that the case should be treated by amputation mainly on account of the radiological and histological evidence of extensive involvement of soft tissues.

On April 4 the arm was amputated by Mr. Brathwaite through the lower third of the humerus. The axillary glands were not touched, as the patient was rather shocked by the main operation. Recovery was unimpaired, and when the patient was discharged from hospital it was observed that the axillary glands were no longer palpable. A longitudinal section through the forearm with the limb in the midway position between supination and pronation (Fig. 261), shows the distal half of the radius to be replaced by a large, solid, pyriform tumour, measuring 2½ in long by 1½ in broad. Its cut surface is pale

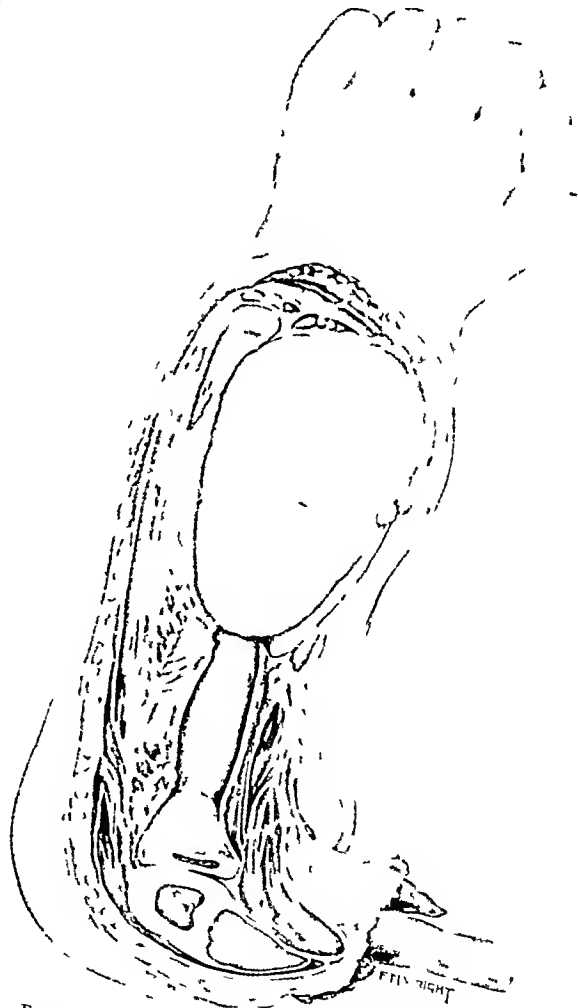


FIG. 261—Myeloid sarcoma of the distal half of the left radius shown on anteroposterior section, after amputation of the arm. The tumour is white throughout and shows neither gross necrosis nor hemorrhage, nor cystic change. (Drawing by Miss Ithel Wright)

throughout, being to all intents and purposes white, with a few small yellowish patches here and there, and two greyish, slightly translucent areas near the centre. It is fairly sharply outlined at the margin, but shows evidence of infiltration of the surrounding tissues. On its radial side it has caused great pressure on the muscles, which are thinned out and pale in consequence. It has also partially surrounded one of the flexor tendons, which lies in a deep groove in the growth. On the opposite side the tumour is in direct contact with the lower half of the ulna, which is slightly distorted in consequence.

HISTOLOGY (Fig 262).—The microscopic structure is that of a typical myeloid sarcoma, but without the usual areas of congestion and hemorrhage. The preponderating tissue consists of a mixed- and spindle cell ground work, with innumerable multinucleated giant cells of osteoclast type. Scattered throughout this are many small, comparatively acellular areas of dense fibrous tissue, containing few or no giant cells. The two centrally situated greyish, translucent areas mentioned in the naked eye description consist solely of fibrous tissue cellular around the vessels, mucoid, comparatively acellular, and in parts even necrotic, away from them. In these fibrous areas, only on occasional small, shrunken giant cell is seen. Undoubtedly the most cellular portions of the tumour are at the periphery, and it is here that the giant cells are, if anything, most abundant. In the highly cellular areas, the mixed cell ground work of the tumour

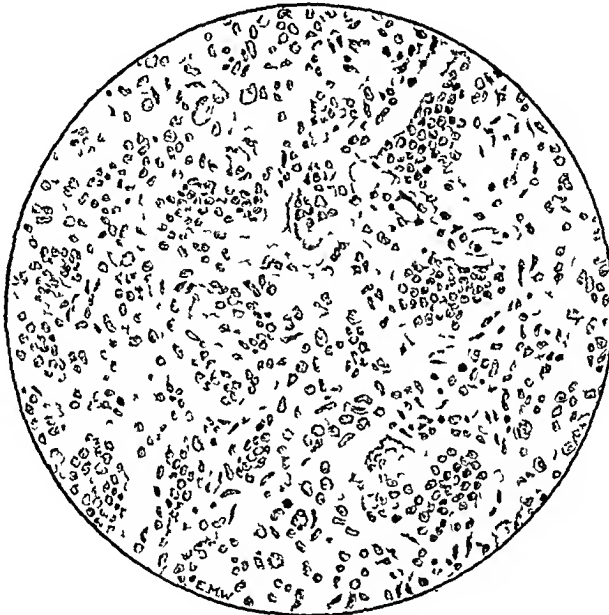


FIG 262.—Drawing of a typical portion of the tumour showing a mixed cell stroma and many large osteoclast like giant cells. (Miss Ethel Wright)

is in a state of active proliferation, mitotic figures being present in abundance. Both cells and nuclei vary greatly in size and shape, and the whole appearance is that of a mixed cell (preponderatingly spindle cell) sarcoma.

The giant cells have very numerous, small, uniform nuclei, usually centrally situated and often arranged in a whorl. They do not show any mitotic figures. The cytoplasm is homogeneous or granular, and stains more deeply than that of the other cells.

Over extensive areas of the surface of the tumour the bony shell has been completely destroyed. Occasionally a tiny spicule of bone is seen embedded in the growth, otherwise there is no evidence of ossification. Wherever the bony covering is lacking, infiltration of the surrounding soft tissues—muscle, adipose tissue, etc., may be seen, and in places there are appearances suggestive of penetration of blood vessels by the giant cells.

The chief interest of this case lies in the colour of the growth. Sarcomas of bone, whether endosteal or periosteal, which are white in colour, are almost invariably highly malignant and most surgeons, on finding such a tumour at operation, would probably be prepared to amputate forthwith. Amputation was carried out in the present instance, not because of any doubt on the part of the surgeon as to the comparatively benign nature

of the growth, but on account of the fact that the tumour had completely burst its bony capsule over a large area, and was actively invading the surrounding soft parts. In spite of its large size and highly cellular character, the growth is white and succulent throughout. It is neither hemorrhagic nor cystic, yet its histological characters are quite unmistakably those of a myeloid sarcoma or so called myeloma. Anything more unlike 'hemorrhagic granulation tissue' it would be difficult to imagine, and the case is a striking commentary on the hypothesis advanced by certain American authors that myeloid sarcoma is neither more nor less than a 'chronic hemorrhagic osteomyelitis'.

A second point of interest is the presence of enlarged axillary glands on the same side as the tumour. It is unfortunate that the condition of the patient did not permit of their removal at the time of operation, as a histological investigation would have been most desirable. Enlargement of associated lymph glands is by no means rare in cases of myeloid sarcoma, but, so far, no conclusive evidence has been produced that this is due to metastatic deposits. Jonathan Hutchinson,² in his *Illustrations of Clinical Surgery*, figures a popliteal lymph gland from a case of myeloid tumour of the tibia, in which 'the peculiar tints of the gland growth were very striking, and closely resembled those of the parent tumour.' No record of microscopic examination had been preserved, however. Hutchinson adds, "I do not recollect to have ever seen the glandular growths in well-marked myeloid disease prove troublesome. It is very exceptional for the glands to enlarge at all, and when they do so it is only to a moderate extent, and with apparently a good possibility of spontaneous retrocession."

I would suggest that lymph-gland enlargement in myeloid sarcoma may be due to the absorption of blood and disintegration products of the tumour by the lymphatics, and in certain cases it is conceivable that this might produce the appearances figured by Mr Hutchinson.

In view of the known behaviour of sarcoma generally, one might reasonably anticipate that dissemination of myeloid sarcoma, if and when it does occur, would be by way of the blood-stream and not by the lymphatics. That a tumour of this kind is capable of penetrating and spreading along the veins was conclusively shown in a case reported by Dr Bristowe³ as long ago as 1855, where a number of large veins were filled with tumour tissue having naked-eye and microscopic characters identical with those of the primary growth. In spite of the fact that tumour-filled veins were present in the amputation flaps, the operation wound ultimately healed satisfactorily, although the flaps sloughed in the first instance. It may be that this accident was responsible for the satisfactory healing which followed. The after-history is not given.

SUMMARY

A case of myeloid sarcoma of the lower end of the radius is reported in which the tumour, measuring 2½ in. by 1½ in., was white throughout. The patient was a small girl of six years, and the swelling had first been noticed three years before. As the tumour had burst through its bony capsule over a large area and was extensively invading the soft tissues, treatment by amputation was decided on, and carried out. Histologically, the growth was a typical myeloid sarcoma (myeloma). The axillary glands on the affected side were enlarged, but had completely subsided by the time the patient was discharged from hospital.

I have much pleasure in acknowledging Mr Braithwaite's kindness in acceding me every facility for the investigation of this case, and my thanks are due also to Dr Leo A Rowden for the accompanying radiograph.

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- HUTCHINSON JONATHAN *Illustrations of Clinical Surgery*, London, 1875, Plate LVI and pp. 77-80.
- ³ BRISTOWE J S, 'Myeloid Tumour of the Humerus', *Trans Path Soc Lond*, 1856, vol. 35, 1.

SOME CYSTOSCOPIC APPEARANCES IN TUBERCULOSIS OF THE URINARY TRACT.

By W GIRLING BALL, LONDON

This paper is intended to illustrate some of the pathological changes which may be observed in the bladder in cases of genito-urinary tuberculosis.

It is now a generally accepted fact that in about 80 to 90 per cent of cases of vesical tuberculosis the primary focus of infection is situated in the kidney, this figure being placed even higher by some observers, the genitalia in the male provide the remainder. Primary tuberculosis of the bladder is very rare, its existence being denied by many observers. It only too frequently happens that there are no renal symptoms in even advanced cases of tuberculous disease of the kidney, whereas the vesical symptoms may be very prominent and lead the clinician to think that the disease is limited to the bladder. Cystoscopy—especially when combined with the use of ureteric catheterization—however, has taught us the true nature of these cases, and has made more obvious the rarity of a primary lesion of the bladder, owing to the greater ability to demonstrate the existence of renal lesions which could not be demonstrated prior to the introduction of this method of investigation. Cases of primary infection have been recorded, however, and one has come under my observation which was proved up to the hilt.*

Renal tuberculosis is a slowly progressive disease which unfortunately may not give rise to symptoms of a sufficiently definite character to cause the affected person to seek advice until, it may be, considerable destruction of the affected organ has taken place, in fact, the involvement of the bladder, with coincident symptoms, may be the first indication of this serious malady. Symptoms indicating involvement of the kidney are of slow development and may be absent altogether, even when that organ has been completely destroyed, dysuria associated with frequent micturition at night as well as by day being commonly the first evidence of disease.

With primary infection of the genitalia, on the other hand, the involvement of the testicle soon attracts attention owing to the external situation of that organ, and it is rare for bladder symptoms to develop at an early stage of the symptomatic history of the disease.

Ability to recognize the appearances seen in the bladder by cystoscopic examination is therefore of great importance in the diagnosis of renal tuberculosis, more especially in demonstrating which kidney is at fault, as in 80 to 90 per cent of cases, so it is stated, one kidney only is affected in the early stages.

It is frequently, from the technical point of view, a difficult matter to carry out a satisfactory cystoscopic examination in this condition. Prior to the appearance of tuberculous lesions in the bladder mucosa little difficulty presents itself, but when the latter exist, then, owing either to a spasm in the early stages or to infiltration of the muscle of the bladder wall in the later stages, it is often impossible to make a thorough investigation unless the patient is under a general anæsthetic, the distention of the bladder with fluid in sufficient amount causing severe pain. It is my practice, whenever a tuberculous infection is suspected, to adopt this procedure, even then, extreme care must be taken

* Shown by Mr Jocelyn Swan at the Cancer Hospital at a recent meeting of the Urological Section of the Royal Society of Medicine.

to avoid over distention of the bladder, otherwise hæmorrhage may be caused and irreparable damage supervene such is the lighting up of a latent lesion or the introduction of a secondary infection, results easily induced by even slight trauma.

There is, moreover, a further difficulty in diagnosis from the pathological aspect namely, that lesions may have assumed such characters, especially in the presence of secondary infection with other bacteria, as to render them indistinguishable from those associated with other forms of cystitis even in the absence of the latter, the bladder may have become so extensively involved in the tuberculous process as to make it impossible to identify the original site of the bladder lesion, which may be the only clue indicating its possible origin from the kidney. It is true that rest in bed free diuresis, and the use of urinary antiseptics will frequently so improve such conditions, even in advanced cases, as to make a diagnosis possible, but such attempts, even when successful, always cause delay. For this reason it is an extremely important matter that in early investigation of the bladder should be carried out in all suspected cases of infection, firstly in order to define the existence of any abnormality in the effluxes from the ureteric orifice or in the bladder mucosa, and secondly to recognize the position of such changes in order to obtain an indication of the site of the primary lesion. The latter point requires emphasizing for the finding of tubercle bacilli in urine containing blood or pus, with symptoms of cystitis, only indicates the presence of urinary tuberculosis it offers no suggestion as to the requisite treatment. Thus the cystoscope alone can give in the absence of localizing symptoms, which as above stated may be completely absent, even if the latter are present, confirmation is always necessary.

Some writers state that it is unwise to carry out cystoscopic examinations in cases of tuberculous cystitis owing to the liability of causing further damage. It is agreed that it is unwise to employ this method of investigation during the acute symptoms of bladder infection, or more often than is necessary in the chronic stage, but it is obviously important that an exact location of the bladder lesions should be made as soon as possible, in order that the correct treatment may be determined upon. This information cannot be obtained without a cystoscopic examination, which should therefore be insisted on.

The illustrations here shown are taken from cases of unilateral renal tuberculosis (with the exception of Figs 272 and 273), and indicate the character of the lesions met with in the bladder in the early stages of its involvement.

The earliest cystoscopic appearance observed is the discharge of blood, pus, or caseous material from the ureteric orifice, or from both orifices if both kidneys are affected. These effluxes, in the absence of lesions in the vesical mucosa, are demonstrable only when it is the custom of the observer to carry out routine cystoscopic examinations in all cases of hæmaturia and pyuria, for their existence is often unassociated with other symptoms or signs. Owing to the fact that the discharges are frequently intermittent, especially in the case of hæmaturia, which is a relatively uncommon symptom of the disease, and are dependent on the pathological changes taking place in the kidneys, several observations may be required in order to demonstrate their source, the period of their discharge is obviously the only suitable time at which the case should be investigated by this method. Even then the discharge may be so small in amount that it may not be distinguishable to the naked eye, under these circumstances the origin of the blood or pus can only be proved by bilateral ureteric catheterization and subsequent examination of the urine collected. At this stage the bladder mucosa frequently shows no change at all, which, from the therapeutic point of view, is the ideal period at which to make a diagnosis. As, however, hæmaturia and pyuria are the signs of diseases other than tubercle, it may not be possible, in the absence of bladder lesions and a failure to demonstrate the presence of tubercle bacilli, to make a diagnosis of tuberculosis, nevertheless, the persistence of the hæmaturia or pyuria thus observed by cystoscopy as coming from one kidney is indicative of its origin, and may, by a process of exclusion, fully justify an exploration of the affected organ.

Fig 263 illustrates the typical appearance of a discharge of blood from the ureteric orifice. *Fig 264* illustrates the discharge of inspissated pus

The case shown in *Fig 264* was of interest, for, on examination, the portion of the bladder wall on which the ureteric orifice was mounted was bulging into the bladder for a considerable distance, the orifice itself, situated at its apex, being plugged with inspissated pus or crassous material. The discharge of the pus was only obtained by passing a ureteric catheter into the orifice, when it ran freely. The patient had a very large pyonephrosis which drained into the bladder after this manoeuvre. It is noticeable that there was very little change in the surrounding bladder wall, which was of the same appearance elsewhere and showed no abnormal lesions. At the subsequent operation it was found that the kidney of that side was completely destroyed. The patient alleged that he had not had any symptoms until fourteen days previously.

The picture in this case may be described as an extreme appearance of pre-vesical involvement, and is very infrequently seen, the more common condition is to find that the discharge of pus is small in amount and difficult to observe cystoscopically, and its presence may be demonstrable only by ureteric catheterization. The two pictures, however, serve to illustrate the desirability of early cystoscopic examination in order to establish the origin of pyuria or hematuria.

Let us turn now to the changes which take place in the bladder wall itself. These vary largely with the stage of infection. Broadly speaking, the degree of involvement of



FIG 263



FIG 264

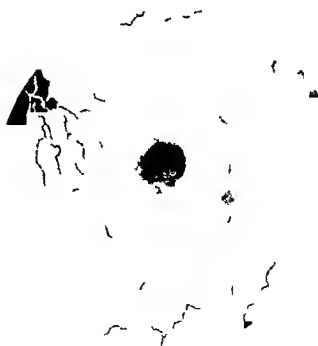


FIG 265

the bladder wall is an indication of the extent of the disease in the kidney. It is said that patients often exhibit symptoms of vesical irritation prior to the appearance of bladder lesions, such being attributed to reflex action. In my experience this is not the case, there is usually some change occurring when these symptoms commence, although it does not necessarily exhibit the characteristics of a tuberculous lesion. It is conceivable, how-

ever, that lesions of the lower end of the ureter may give rise to vesical symptoms without actual involvement of the bladder mucosa.

The earliest changes are usually found in near relationship to the primary focus of infection—i.e., around the ureteric orifice in cases of renal origin, and over the vesicle or prostate when the genital organs are the offenders. Sometimes, however, the first bladder lesions make their appearance in other areas of that structure, leaving the sites above mentioned free.

In cases of renal tuberculosis the lips of the vesical orifice of the ureter become swollen, hyperæmic, œdematous, and may have bullæ around them. These bullæ, which are semitranslucent in appearance, may become heaped up on each other (bullous œdema), so as to hide the site of the orifice, and the œdema may spread on to the base of the bladder and trigone. This condition is considered by some to be pathognomonic of tuberculous infection, but there is no doubt that it may be found in other chronic infective conditions. In fact, the appearances just mentioned only indicate the existence of an infection of the renal tissues. It is difficult to obtain a suitable illustration of the congested state of the ureteric orifice (shown in *Fig 265*), but *Fig 266* illustrates most beautifully the condition of bullous œdema.



FIG 266

When this patient was first seen, he gave a history of two years' pyuria, with the symptoms of cystitis, and a loss of 2 stone in weight. He was thought to be suffering from tuberculous disease of the urinary tract, although after repeated investigation the bacilli had not been demonstrated.



FIG 267



FIG 268

The bacteriological examination always showed the presence of *B. coli communis* and streptococci. Both of the seminal vesicles were hard and nodular, as also was the prostate. The cystoscopic examination did not give any satisfactory clue as to the origin of the condition, showing the bladder to be in a condition of severe, diffuse, chronic cystitis, with the suggestion of most advanced changes in the region of the left ureteric orifice. There was no obvious ulceration.

the mucosa being tremendously œdematous and covered with shreds of thick mucus pus which could not be washed away. There were no renal symptoms.

The patient was treated by rest in bed, free diuresis, and antiseptics, and as he preferred to have this carried out in his own home, he did not come under observation again for six months. He was then very much better in his general condition, and his vesical symptoms had much improved, renal symptoms were still absent. The appearances in his bladder had completely altered, and illustrate the advantage of improving the condition of cystitis prior to coming to a definite conclusion as to the nature of the lesion. The condition shown in the figure was the only abnormality found in the bladder. The left ureteric orifice, discharging pus, could just be seen, surrounded by bullous œdema. The right ureteric orifice was natural in appearance. On bacteriological examination of the urine the *B. coli communis* only was found, and tubercle bacilli were still absent.

Nephrectomy was performed, and the cortex of the kidney was found to be the site of multiple large chronic abscesses, which on microscopic section failed to demonstrate the characters of a tuberculous infection, though the lesions appeared to be of that type.

Although this case was one of chronic *B. coli* and streptococcal infection of the kidney, the appearances seen around the ureteric orifice serve well to illustrate the changes which may be seen in cases of chronic tuberculous nephritis.

The swelling and congestion tend to spread over the mucosa of the bladder base

around the orifice of the ureter. The appearance of a delicate network of dilated blood-vessels arranged in a flame-like fashion in this region, or even more extensive sub-mucous hæmorrhage, is characteristic (Figs 265, 267, 268). All the patients presenting the above appearances had advanced tuberculosis in the corresponding kidney, as was proved by subsequent nephrectomy. In the first case (Fig 265) the renal tissue had been completely destroyed and a large pyonephrosis had formed. The ureteric orifice in two cases (Fig 265 and Fig 267) illustrates the typical 'golf-hole' appearance so commonly associated with an inactive kidney or a blocked ureter. In the second case (Fig 267) it is seen that the hæmorrhagic patches are lying above the ureteric orifice, behind the interureteric bar, more or less along the line of the lower end of the ureter, this is a common site for the



FIG 269.

appearance of early tuberculous lesions, and very suggestive of a direct infection of the bladder mucosa through its wall from lesions in the ureter.

The changes so far described are identical with those associated with other forms of chronic inflammation, and must not be regarded as pathognomonic of a tuberculous infection.

Miliary tubercles, the characteristic lesions of tuberculosis, are not seen so commonly as might be expected, when they are present, however, they possess appearances analogous to similar lesions seen in other mucous membranes, first as grey, pearl-like nodules with a smooth surface, and later as minute yellow areas of caseation, the size of a pin's head (Fig 269), usually multiple, slightly raised from the surface, with clear cut margins, and with a tendency to increase in size and to coalesce. There is a small zone of hyperæmia around each tubercle, which, if the latter are large in number, tends to cause congestion of a considerable area of the bladder mucosa. The tubercles are usually situated around the ureteric orifice, but may be found at some point a little distance from it. In the early stages of vesical infection the rest of the bladder mucosa maintains its normal appearance. The same lesions may be seen situated over the vesicle or prostate when these organs are the primary foci of infection.

Eventually these tubercles break down and leave small shallow ulcers with a sharply defined, slightly raised, undermined edge, often bright red in colour (*Fig 270*), usually a little ragged owing to the adhesions of mucus, with a shallow necrotic base covered with a yellow slough, which later clears away and leaves unhealthy pinkish-blue granulations. When several tubercles fuse together these ulcers may be of quite a considerable size exhibiting rather irregular edges due to their fusion, but otherwise having similar characters. As the disease advances, these lesions become more numerous and spread to a distance in the bladder wall far from the orifice of the originally infected ureter. The mucosa surrounding these ulcers varies considerably, sometimes it is natural in appearance, at others there is a deep zone of congestion, especially when many ulcers are present indicating a more active stage of the disease. Yet again an appearance of healing may be observed at the edge of an ulcer while it spreads in another direction they may even heal completely, leaving weak cicatrices which are very prone to break down again this is characteristic of a tuberculous lesion. At other times the ulcer may be deeply excavated, especially when the primary origin has been in the seminal vesicle or the prostate, the edges of such an ulcer are usually irregular and undermined, although



FIG 270



FIG 271

not markedly raised from the surface of the bladder mucosa, the base is frequently occupied by blood-clot in the recent state or a slough in the later stages.

In rare instances masses may be seen, sometimes having a papillomatous surface, which may be spoken of as 'tuberculous granulomata'.

The appearances seen in *Fig 270* are typical the ureteric orifice with a swollen margin is seen at the lower edge of the picture, extending in a backward direction from this along the line of the ureter can be seen a number of small shallow ulcers of characteristic appearance. *Fig 271* represents the left ureteric orifice of the same case, the two pictures demonstrate the marked differences in appearance which may be seen in the same bladder, the mucosa around the lesions being deeply congested, whereas that on the normal side is quite natural.

This patient was a young girl who had never had any renal symptoms at all, and who for four months before she had come under observation was suffering from increased frequency of micturition. No tubercle bacilli were found in the urine after repeated examination. Nephrectomy was performed, and two caseous tuberculous foci discharging into the pelvis were found in the kidney, the renal pelvis being studded with tubercles. The bladder exhibited the changes shown. She made a complete recovery, the bladder at the present time exhibiting normal cystoscopic appearances with the exception of a white scar at the site of the ulceration (*Fig 274*).

Fig 272 represents the appearances of a single ulcer found in the bladder of a man complaining of vesical symptoms who had had his right testicle removed for tuberculous disease some months previously, and who at the time this picture was taken had a hard, nodular seminal vesicle underlying the site of the ulcer. The clean cut margins with the

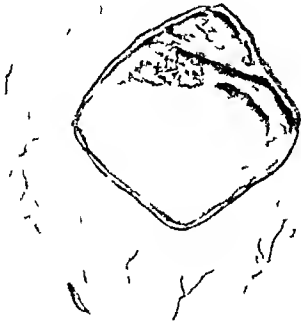


FIG 272

pale reddish base, with practically no undermining of the edges give a characteristic impression, more especially seen in the near view (Fig 273), which demonstrates the pinkish-blue appearance of the granulations. Attempts were made to prove that the kidneys were infected, but failed, as might be expected from the normal appearance of the meterie orifices.



FIG 273

Tuberculous cystitis has a tendency to remain localized until other infective bacteria take part in the process. With the onset of a secondary infection, the changes already described become less obvious. The mucosa of the bladder wall between the lesions which up to this period retains a more or less normal appearance, now exhibits changes usually associated with a chronic cystitis of pyogenic origin, which masks the characteristic appearances of a tuberculous lesion. It is this change which so frequently makes it difficult to arrive at a correct diagnosis.

In the stage of healing, scars form in the bladder at the site of ulcers (Fig 274), these, though having the characters of scars elsewhere, have a ready tendency to break down and re-ulcerate.

The 'golf hole' meterie orifice through which there is no discharge, with the orifice drawn up to a higher level than that of the opposite side, is the common appearance seen in the case of a long standing active or calcified tuberculous kidney. It is no doubt due to infiltration of the ureteric

wall with inflammatory material, which renders its lips rigid and non contracting and apparently protruding further into the bladder cavity than normal. The shortened and thickened ureter can often be seen producing a ridge (Fig 275) in the bladder wall on the renal side of the ureteric orifice. The picture shown came from a case of advanced tuberculous disease of the kidney which had become secondarily infected with *B. coli communis*. It is interesting to note the absence of cystitis in this case despite the presence of symptoms of renal disease extending over a period of seven months.

In the long-standing cases the bladder wall usually becomes contracted and thickened owing to widespread infiltration, masses of caseous material may even be deposited in its structure.

Such are some of the appearances which may be observed but, as has been stated, it is often by no means easy to be sure that lesions seen in the bladder are of tuberculous origin. The history of the condition, with typical symptoms, especially if tubercle bacilli can be demonstrated in the urinary deposit either by film or cultural preparation, and more certainly by guinea-pig inoculation, will, as a rule, make the diagnosis of urinary tuberculosis certain, if the bacilli are not found on the first investigation, repeated examinations may lead to a successful result,

if sufficient care is taken they can be found in the majority of cases. But if these classical symptoms and signs fail to indicate the nature of the condition, cystoscopic examination may be the only means of arriving at a diagnosis, in every case it is the only means of indicating which kidney is the primary source of the vesical lesions, and the only method combined with ureteric catheterization, of defining a unilateral infection. These investigations demand the greatest care and patience, but they are worth it when good results are obtainable. It is clear that the diagnosis should be made at the earliest possible moment after the onset in order to obtain the best results, and this may only be achieved by the use of the cystoscope.



FIG 275

I am greatly indebted to Mr W Thornton Shiells for the care and skill with which he has represented the cystoscopic appearances.

AN ACORMOUS EMBRYOMA, CONSISTING OF A HYDROCEPHALIC FETAL HEAD CONTAINED WITHIN AN OVARIAN CYST, IN A CHILD $2\frac{1}{2}$ YEARS OF AGE. OVARIOTOMY

By C E SHATTOCK, LONDON

THE diseased ovary in this case was removed by the author from a child $2\frac{1}{2}$ years of age who had suffered from indefinite abdominal symptoms for six weeks. On examination, a firm, clearly defined freely movable tumour was palpable in the mid-line between the pubes and umbilicus. The parents and three other children were healthy.

The specimen, which may be described somewhat fully for the sake of any future reference that may be made to it, is represented of the natural size in the accompanying figure (*Fig. 276*)

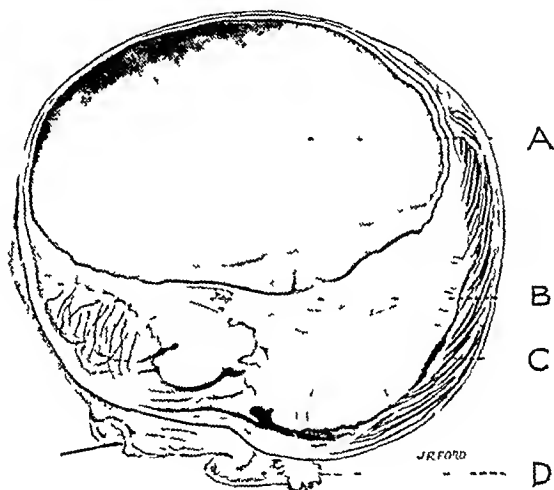


FIG. 276.—A sagittal section of the acormous or trunkless ovarian embryoma described in the text. Most externally is seen the thin-walled ovarian cyst. This encloses the embryo, which is represented solely by a hydrocephalic head. The mass of dark hair on the right-hand side growing from the skin is apparently that of the scalp. At the bottom of the figure and connected with the exterior of the ovarian cyst, there is a normal Fallopian tube. A, Interior of the hydrocephalic brain; B, Bone representing a basis cranii; C, Thick, dark hair growing from a scalp; D, Fallopian tube normally attached to the exterior of the distended ovary. The specimen is now in the Museum of the Royal College of Surgeons, London [General Pathology Cysts] (Natural size).

The specimen consists of half of the left ovary of a child, enlarged by the growth of an embryoma, which is composed solely of the head of a hydrocephalic foetus and completely fills a cyst in the ovary. The soft, easily separable hydrocephalic brain measures $5\frac{1}{2} \times 3$ cm in chief diameters and is lined with ependyma through which the subjacent vessels are visible. Below the middle of the distended brain there is an elongated piece of cancellous bone closed in with a layer of compact bone, which may be taken as the basis cranii. Below the bone, and extending behind it as far as the skin, and beneath the brain, there is a triangular mass of young connective tissue and fat, in which microscopic examination demonstrates also the presence of a few islets of cartilage and a compact, ill-defined mass of well-developed, intersecting bundles of unstriated muscle fibres. There is no clue as to what these structures morphologically represent. Lastly, there are included groups of ganglia furnished with large, typical nerve cells. The skin over the triangular mass of connective tissue already referred to is thickly covered with somewhat stiff dark hair embedded in sebum, the hair lies immediately against the inner surface of the enveloping

cyst, which is so thin that the former is obvious through it. The connection between the embryoma and the wall of the cyst is limited to a strand of connective tissue which passes from the lower aspect of the trigulum into the opposite side of the cyst. In front of this connection, the solid tissue of the trigulum is confined to a small oval sessile process projecting into the cavity of the cyst below (see Fig 276) and, more interiorly, to a stretch of skin bearing hairs embedded in sebum. The proper wall of the cyst, the skin, and cerebral substance are traceable from the base, becoming more and more attenuated, to the vertex. The epithelial investment of the oval mass projecting into the cyst below the base of the skull affords matter for consideration. For whilst it is covered mostly with ordinary epidermis and furnished with hairs and sebaceous glands, the epithelium for a short distance is columnar celled and ciliated, the one being continued into the other. Whether this represents a mucocutaneous junction, it is impossible to say, there is no pertaining unstippled muscle. The cavity of the hydrocephalic brain is subdivided into an anterior and posterior fossa by means of a subjacent ridge continuous with the central process of bone before mentioned, the general disposition corresponding with that of the interior of the base of the skull.

This specimen appears to be unique. The embryoma is represented by a head without either trunk or limbs.

Such an acormous or trunkless condition is recognized in the case of teratological uterine monsters, and although there is no example of it in the extensive Teratological Collection at the Royal College of Surgeons, London, full references to twelve instances are given in Alshfeld's exhaustive *Missbildungen des Menschen*, 1880, and it is the condition present in what is one of the most remarkable forms of double monster yet observed, viz, that of Hunter (No 166, *Teratological Series, Roy Coll Surgeons*), which was described by Sir Everard Home in the *Philosophical Transactions*, Vol LXX, p 296. This preparation is the skull of an Indian child, to the anterior fontanelle of which there is attached a second inversed skull, almost equally large, the faces of the two being turned in opposite directions. During life the second head was surmounted by a short neck which was soft at the age of two years, and quite firm and cartilaginous at four. The brains were found, after death, to be distinct, but their dura mater was coherent. The facial movements of the upper head were reflex, and not controlled by the feelings of the child, the eyelids were usually open, even when the child was asleep.

Hunter's specimen is explicable as a double monster of which the second head represents a foetus, all the parts of the latter, except the head, having aborted so as to result in an acormous parasite.

The most common form of double monster is that in which the union is back to back, in the sacral region—pygopagus. Of head to head fusion—craniopagus—there is one example only in the College Museum (No 165), the twins are immature, and in other ways abnormal (*Lancet*, 1876, Aug 26). According to the site of union, the condition is subdivided into craniopagus frontalis, craniopagus occipitalis, and craniopagus parietalis. Hunter's specimen belongs to the 'parasitic' sub-variety, the second individual being a mere appendage to the host. Three other specimens of parasitic craniopagus were discovered by Alshfeld in teratological literature.

The specimen here recorded may be regarded as the complement of another, also in the College of Surgeons, which was fully described by Professor S G Shattock (*Path Soc Trans*, Vol LXX, p 267) in a paper devoted to the so called dermoid cysts of the ovary, in which exists the embryoma may have aborted and dwindled to a patch of piliferous skin overlying an eminence of subcutaneous fat. This specimen (No 1228 I) comprises a trunk, a caelomic cavity containing a blind loop of intestine, and processes representing limbs but no head. The embryoma is acephalous, and not—as in the specimen under consideration—acormous. What is physiologically remarkable, moreover, is that in the mass of fat of which the trunk is largely composed, there is a well-formed pelvis and that from the skin over the pubes there has grown a well-pronounced tuft of hair. This obviously marks the pubescence of the intra-ovarian parasite, which must be explained by the recess of endocrine from the maternal blood to the embryoma when the bearer herself attained puberty, seeing that there are no sexual glands in the parasite, and that the bearer was an adult (loc cit).

The theory propounded in the paper referred to is at least one possible way of viewing

the origin of intra ovarian teratomata, and may be very briefly referred to. It is that one or more of the primordial germ cells in the developing embryo are, at a very early stage, 'fertilized' by spermatozoa left over after the normal physiological fertilization which gives rise to the main embryo. This later secondary and spurious fertilization gives rise to the intra-ovarian monster which develops to a varying extent within the genital gland of the foetal ovum by the extrusion of polar globules. The 'fertilizing' spermatozoon would act mechanically only, and stimulate the ovarian cell to start subdividing, as may be effected mechanically in lower forms of life, and in the ova of the frog by puncture of the unfertilized ova.

Lastly, there is a further point of interest in the coloration of the hair. The hair of the scalp of the child is pale fawn, that of the scalp of the father and the mother is brownish black, and equally so, that of the scalp of the embryoma is brownish black, but not so deep as that of the parents. Now double monsters (misdeveloped uniovular or homologous twins) are not only of the same sex, but the hair of the scalp is of the same colour. The coloration of the hair of the ovarian teratomata (so-called dermoid cysts) may not, in fact, correspond with that of the host. This favours the hypothesis that the embryoma is due to the introduction of a 'fertilizing' agent which gains access to the embryonic ovary, rather than that the embryoma is an abnormal product of the developing embryo itself.

The name 'epiembyrogenesis' given to the theory, so to say, was devised to indicate that one embryo (the intra ovarian) is produced 'on the top' of another.

STUDIES IN GALL-BLADDER PATHOLOGY

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I INTRODUCTORY

THE pathological problems presented by that profitable stone-quarry, the gall-bladder, are numerous and varied. What is the route of infection? Is it hæmatogenous, lymphogenous, or by the bile-duct? How are stones formed? What is the explanation of the symptoms of gall-bladder dyspepsia? What is the relation of cholesterol to gall-bladder disease? What is the connection between hepatitis and cholecystitis? What is the meaning of the strawberry gall bladder? And, finally, what is the function of the gall-bladder? In the present communication it is only possible to touch upon one or two of these.

In the past, far too much stress has been laid upon the presence of calculi. Monographs have been written on gall stones and the pathological alterations of the bile. But calculi are incidental, not essential, to gall-bladder disease. Their presence may lead to dramatic symptoms which compel attention both on the part of the patient and his physician, but they have distracted attention from the essential problem, namely, that of the gall-bladder itself.

When the surgeon opens the abdomen in search of a diseased gall-bladder he may find one of three conditions —

- 1 Acute inflammation, usually accompanied by the presence of calculi
- 2 Chronic inflammation, with or without calculi
- 3 The gall-bladder may appear little if at all altered (though usually its normal bluish semi-translucent appearance is lost) but when it is opened the mucosa is seen to be dotted with minute yellow spots, a condition to which the name of strawberry gall-bladder was first given by McCarty.¹ A study of this peculiar condition may throw some light on the general question of gall bladder pathology.

The Strawberry Gall bladder—The term strawberry gall-bladder was used by McCarty because of the resemblance which the tiny yellow specks, scattered over a reddish background, bore to a ripe strawberry. At first McCarty considered that the appearance was to be explained by a desquamation of the epithelium covering the villi, thereby allowing the underlying connective tissue to become stained with bile. Later he recognized that

the yellow material must be lipoid in nature, for it stained red with Scharlach R, more-over the loss of epithelium was merely due to trauma

My own studies upon the nature of this lipoid were at first purely histological, but as the scope of the investigation widened, it was found necessary to employ chemical and experimental methods, and finally excursions had to be made into the realm of comparative anatomy

II METHODS OF INVESTIGATION

The first point to be determined was the nature of the lipoid. The material, which was obtained absolutely fresh from the operating room, was stained with the usual fat stains, Scharlach R or Sudan III, and with osmic acid. Lorrain Smith's Nile blue sulphate method was used for the detection of fatty acids. In some cases the Weigert-Pal method for myelin was employed. Sometimes the tissues were examined fresh but the best results were obtained with frozen sections cut after preliminary fixation in formalin. Paraffin sections were studied in every case for the finer histological changes. Three of

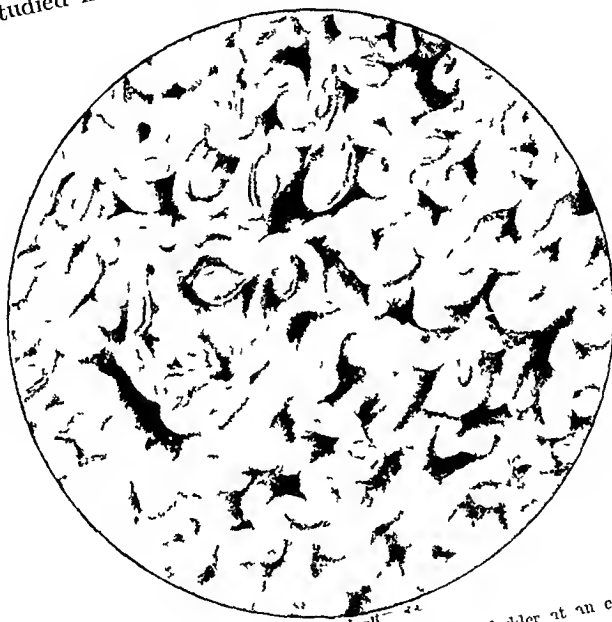


FIG. 277.—Mucous membrane of a strawberry gall bladder at an early stage.
The patches of lipoid are whitish in colour.

the simplest and at the same time the most valuable methods remain to be mentioned. These were the low-power binocular or dissecting microscope, whereby a direct stereoscopic view of the mucosa could be obtained, the polarizing microscope, and the ordinary microscope with the diaphragm closed for the study of the unstained section.

1 The Dissecting Microscope—For the study of many diseased tissues this is an instrument of the greatest value. It gives one an intermediate picture between that of the naked eye and of the microscope slide. The idea one gets of the structure of the gall bladder both in health and disease, is entirely different from that derived from the study of sections.

Moreover, the histological study of the normal gall-bladder is beset with difficulties. I have found autopsy material absolutely useless. Even when the gall bladder is removed as early as three hours after death, degenerative changes of a most serious nature have occurred in the villi, and the epithelium is usually completely desquamated or else indistinguishable. In studying this phenomenon, portions of fresh gall-bladders removed at

operation were kept for varying periods in water, in a moist atmosphere, and in bile. Under these circumstances the changes were not anything like so marked as in the autopsy specimens, but in the case of the tissue kept in bile they were very considerable. The degenerative changes are therefore probably due to the action of the bile on the delicate epithelium.

In text-books of anatomy one reads the statement that the mucous membrane of the normal gall-bladder is thrown into folds. But this conveys little to the mind until the gall-bladder wall is viewed direct under the dissecting binocular microscope. When the fresh gall-bladder, immersed in water, is observed by reflected daylight or, still better, by the brilliant light of an electric arc, the picture is a remarkable and beautiful one. As if one were gazing into the depths of a marine pool at sea-weeds and sea-anemones tall graceful folds and membranes, gossamer-like in their delicacy, can be seen floating in the ambient fluid. The entire inner surface is divided by these membranes into a series of polygonal spaces, and each of these spaces resembles a little courtyard surrounded by high



FIG. 278.—Masses of lipid in the stroma. The surface epithelium is intact. Stained with Scharrlach P.

though delicate walls. In microscopic sections the membranes, cut transversely, appear as villi. They are not true villi, but the term is allowable because of its convenience. It is a striking picture and, as we shall see later one which at once suggests that the idea of the gall-bladder being a mere reservoir is absolutely untenable. Such a highly specialized structure can be for one purpose only, and that purpose is absorption.

In the strawberry gall-bladder the picture is even more wonderful. The lipid in marked cases is seen in the form of dense yellow masses of dull hue. The graceful, fragile, gossamer folds of mucosa are completely altered in appearance, being loaded down by the dense, opaque lipid much as a delicate birch tree might be weighed down by a load of snow. Sometimes the lipid is confined to the summit of the ridges, sometimes, when the ridge is viewed in profile, it can be traced down into the depth of the recesses. In the severe cases the distribution is widespread. In the milder cases it is more patchy, picking out a fold here and there and giving the mucosa the appearance of a mountain ridge retaining only in occasional patch of the winter's snow (Fig 277). Before it is fixed in formalin the lipid can be lifted up by means of a needle in long strings as if it consisted of

molasses The material thus removed can be further studied by methods presently to be described

2 Staining Reactions—The yellow material of the strawberry gall-bladder gives the usual reaction for fat It is soluble in alcohol, ether, and chloroform, so that it cannot be seen in paraffin sections It stains red with Scharlach R and Sudan III, and black with osmic acid In these respects it behaves like ordinary neutral fat, which is an ester of glycerin, and which is sometimes found in large amount in the subserous tissue and in the deeper layers of the fibrous coat of the diseased gall-bladder Although evidently of lipoid nature, it differs from ordinary fat in some most important particulars With Scharlach R it does not take on quite the same brilliant scarlet which neutral fat displays Moreover, it is quite often granular in form, although not infrequently globular (*Fig 278*) but in the latter case the globules are always small and often irregular in form The neutral fat in the deep layers of the wall is in the form of large, spherical globules Nor is the



FIG 279—Deposits of lipoid stained with osmic acid

staining with osmic acid the same as that of ordinary fat The black is not a jet black, often more of a grey (*Fig 279*) and not at all intense

In order to determine the exact nature of the lipoid other methods had to be employed Lipoid material may occur in the body as neutral fat (an ester of the alcohol glycerin with a fatty acid), as free fatty acids, as cholesterol, or as cholesterol ester (an ester of the alcohol cholesterol with a fatty acid) All of these stain red with Scharlach R

Sections of each case were also stained by Lorrain Smith's Nile blue sulphate method This member of the oxazine series of dyes stains neutral fat rose and fatty acids blue The lipoid in the mucosa of the strawberry gall-bladder took on sometimes an intermediate violet tint, sometimes a deep blue, whilst the neutral fat in the subserous tissue stained a definite rose

The question arose as to what colour cholesterol and its compounds would develop with Nile blue The literature contained no answer to this question but the reaction of different dyes with various forms of lipoid was determined in the following manner—

A number of capillary tubes were drawn out, and into each was run first the substance to be tested and then the dye, after which the ends of the tubes were sealed In this manner

triolin, tripalmitin, tristearin (all glycerin or neutral fats), oleic acid, an alcoholic solution of cholesterol, and cholesterol ester made by the addition of cholesterol crystals to a soap solution, were tested against Scharlach R and Nile blue sulphate. With the Nile blue triolin gave a distinct narrow zone of rose at the junction of the two fluids, oleic acid a bluish violet, and cholesterol and cholesterol ester a deep blue. The reaction with Nile blue showed, therefore, that the lipid in the mucosa was certainly not neutral fat, but left undecided the question as to whether it consisted of fatty acids or of cholesterol.

3 The Polarizing Microscope—This microscope, so powerful an instrument in the hands of the crystallographer, has not received the recognition it merits in biological work. For researches upon the lipoids it is absolutely essential, and it is remarkable that this extremely simple method has not come into more general use. All that has to be done is to cut a frozen section of the tissue to be examined, and place it, unstained, between Nicol's prisms which may be attached to any microscope. When the prisms are rotated until they are at right angles to one another all material which is non-refractile, or which is isotropic (singly refractile), disappears, and the field of the microscope becomes perfectly dark. Under the same conditions any material which is anisotropic (doubly refractile) shows up brilliantly white upon the black background.

When an unstained frozen section of a gall-bladder containing lipid is examined with the polarizing microscope the lipid stands out in the most beautiful and brilliant manner, shining with a silvery radiance which is accentuated by the surrounding darkness. Under higher magnification it was seen sometimes to be in the form of granular amorphous masses, but frequently it appeared as innumerable tiny needle-shaped crystals. In some cases a most curious appearance was observed, bright Maltese crosses standing out against a black background. These crosses were never seen actually in the substance of the tissue, they always appeared to be lying free a little distance from the villi.

Neutral fat and fatty acids are invisible under crossed Nicol's prisms. Cholesterol crystals are brilliantly anisotropic, are of characteristic form, and show an exquisite play of colour, in which reds and blues predominate. Cholesterol esters are also markedly anisotropic, but are pure white.

In order to study the ester some cholesterol crystals were added to oleic acid. They remained quite unchanged. When, however, the reaction was made slightly alkaline (as is the case in the bile), ester formation at once commenced. It was found that a solution of soap could conveniently be substituted for the oleic acid. A drop of the solution was placed on a cover-glass, a few crystals of cholesterol were added, the cover-slip was inverted over a hollow ground hanging drop slide, and rimmed round with vaseline to prevent evaporation. The process of esterisation could then be watched under the dissecting microscope. In a very short time the crystals began to lose their sharp outline, and the borders became more and more fuzzy. Gradually those peculiar bodies called myelin figures began to form. These were identical in appearance with the figures which develop when the myelin of the medullary sheath of nerves is placed in water. Long, finger-like processes are pushed out, the ends of which develop first a longitudinal and then a transverse groove, and finally become changed into typical Maltese crosses. The whole is anisotropic. These figures are really crystals in fluid form, they have been called fluid crystals. By the end of twenty-four hours it was found that the cholesterol crystals had completely disappeared, being entirely converted into the ester form. *Under the polarizing microscope the newly formed ester was very similar in appearance to the lipid material of the strawberry gall bladder.* We are now, therefore, in a position to say with a considerable degree of confidence that the yellow material of the strawberry gall-bladder is the ester of cholesterol with a fatty acid.

The polarizing microscope forms a ready and convenient method of examining lipid deposits. It at once distinguishes between the glycerin and the cholesterol series of fats. The uncertainty which sometimes accompanies the staining of fats is not encountered in the method. It has, however, its drawbacks. Precise orientation is difficult owing to the darkness in which the greater part of the section is shrouded, so that it is not possible to be certain if the lipid is in the epithelial cells or in the stroma.

Moreover, all that glitters is not cholesterol. It was soon found, for example, that fibrous tissue appeared quite bright (not brilliant), although muscle was lost in impenetrable darkness. No explanation can be offered why a non-crystalline substance like fibrous tissue should, even though feebly, refract polarized light.

The phenomenon suggests that the method might prove of value in histological work along other lines.

4 Closed Diaphragm—The lipid in the gall-bladder can be demonstrated without the use either of special staining methods or of the polarizing microscope. When a frozen section of a strawberry gall-bladder is examined under an ordinary microscope with the diaphragm well closed, the lipid is seen as dark, almost black masses. Under the high power the acicular crystals can be seen with great distinctness, and the amorphous granular masses can also be made out.

This method, in addition to its simplicity, has the advantage that it obviates the possibility of error due to deposits of such a stain as Scharlach R. When, however, the lipid is small in amount or scattered diffusely, staining methods are required for its detection.

5 Microchemical Reaction—The behaviour of the lipid in the mucosa when viewed under Nicol's prisms suggested very strongly that it must be an ester of cholesterol. It was felt, however, that even more convincing proof might be obtained if one of the chemical reactions for cholesterol could actually be carried out in the tissues. This was manifestly impossible in the case of most of the tests, but it was felt that Moleschott's sulphuric acid reaction might possibly be applied to a section of the tissue.

When concentrated H_2SO_4 , in the proportion of five of the acid to one of water, is added to cholesterol crystals, the latter turn a bright carmine red. The reaction with the ester of cholesterol is not mentioned in the literature, so a film of the ester was prepared, and the concentrated acid added. The resulting colour was a terra-cotta brown. When the reaction was watched under the polarizing microscope an interesting phenomenon was observed. The cholesterol crystals remained as brightly anisotropic as before, but the ester completely lost its power of refraction. The difference could be observed very beautifully when a mixture of the crystals and the ester was used.

The test was then applied to the tissue. A frozen section of a gall-bladder containing lipid was floated on to a slide and allowed to dry; it was gently blotted with filter paper, the acid was then added, and a cover glass applied. The result was highly gratifying. The lipid rapidly became stained: first yellow, then brown, and finally, after a considerable time it acquired a faint tinge of violet or purple. Perhaps the dominant colour should be described as henna rather than brown. It closely resembled the colour produced by the action of the acid on cholesterol ester. Moreover, the anisotropic character of the lipid was completely destroyed by the action of the acid. In short the lipid gave the chemical and physical reactions of cholesterol ester. It was feared that the strong acid would have a disastrous effect upon the tissue, but even the epithelial cells remained undamaged. In the earlier experiments the changes in the tissues were largely obscured by a tremendous evolution of bubbles, due apparently to a union of the concentrated acid with a small amount of water left in the tissue even after blotting with the evolution of gas. It was soon found that this could be overcome by first allowing a gentle stream of acid to flow over the section and after all the bubbles had been produced and removed, then to apply the cover-glass.

Here, then, was a chemical test for cholesterol ester which could be applied to any tissue, and it was felt that a powerful new instrument of investigation had been put in our hand, an instrument which could be applied to other tissues in an investigation into the rôle which cholesterol plays in the animal organism.

The method was at once applied to an organ which is known to be rich in cholesterol, namely, the adrenal cortex, and the result was exactly as had been anticipated. The cortex at once took the brown colour, whereas the medulla remained unstained. The value of the method was thus confirmed in a striking manner.

III AMOUNT OF CHOLESTEROL IN STRAWBERRY GALL-BLADDER

Another problem now presents itself Is the increase in cholesterol in the strawberry gall-bladder real or only apparent? In fatty degeneration of the heart or of the kidney a large amount of fat can be demonstrated by staining methods in the heart muscle fibres and in the epithelial cells of the convoluted tubules But when a quantitative estimation of the fat extracted from the organ by means of a Soxhlet apparatus is made it is often found that the amount is no greater than that in a normal heart or kidney The invisible fat normally present in a combined form has merely become visible owing to the pathological changes induced by the toxic agent The increase in fat is apparent, not real

In order to determine this point, extractions were made of a number of strawberry gall bladders, and of a normal gall-bladder as a control (only one of the latter removed at operation was available) The mucous membrane was separated from the fibrous coat dried, and a weighed portion of each was extracted with a mixture of equal parts of absolute alcohol and ether in a Soxhlet extractor The ethereal extract containing the lipid was evaporated almost to dryness and the residue dissolved in chloroform The amount of cholesterol in the chloroform solution was then estimated by developing the characteristic green colour of the Liebermann-Burchard reaction through the addition of acetic anhydride and concentrated sulphuric acid, comparing in a Bausch and Lomb colorimeter the colour produced with that of a solution of cholesterol of known strength treated in the same way The figures obtained in a series of observations are given in *Table I*

Table I—SHOWING VARYING CHOLESTEROL CONTENT OF THE GALL BLADDER IN DIFFERENT CONDITIONS

TYPE OF GALL-BLADDER	PER CENT CHOLESTEROL BY WEIGHT
Normal	0.51
	1.70
Inflamed (no lipid)	0.36
Strawberry No. 1	60.54
" " 2	46.40
" " 3	31.60
" " 4	50.45
" " 5	51.00
" " 6	41.80

It will be seen that the mucosa of the strawberry gall-bladder contains an enormous amount of cholesterol compared with that of the normal organ The increase is therefore real and not merely apparent It is interesting to note that the fibrous coat shows little or no change

IV DISTRIBUTION OF THE LIPID

The discussion so far has been confined to the strawberry gall-bladder, to the gall-bladder, that is, in which the lipid is visible to the naked eye The material for the present part of the investigation consists of the gall-bladders removed during 1921 and the first half of 1922, 100 in number All of these were examined for lipid by the methods already described It was soon discovered that lipid may be present although none is visible to the naked eye or even under the low-power binocular microscope It was found in 52 cases out of the 100, in only 10 of these was the lipid discernible by the naked eye

The term 'strawberry gall bladder' is sometimes used as if it were a definite pathological entity such as acute cholecystitis From the above remarks it is evident that this is not so Under certain pathological conditions, presently to be discussed, an ester of cholesterol is formed in the mucosa of the gall-bladder When this formation is so marked in degree that the lipid becomes visible to the naked eye, the condition of strawberry gall bladder is present It is all a question of degree The term 'lipid gall-bladder' is suggested this would include both the strawberry gall-bladder and those gall-bladders in which the lipid is not visible to the naked eye

The lipoid is usually scattered over the entire surface of the mucosa. Not all the ridges are involved, nor is every part of a ridge, the distribution is essentially discrete. Occasionally only one portion of the gall-bladder is involved. In one very instructive case (Table II) the organ could be divided into two portions of unequal size. About one-third, including the neck, was comparatively slightly thickened, the ridges were tall, thin, and normal-looking, but there were numerous large deposits of lipoid. The other two-

Table II—SHOWING HOW THE CHOLESTEROL CONTENT MAY VARY IN DIFFERENT PARTS OF THE SAME GALL-BLADDER

Part showing advanced inflammatory changes
Part showing slight inflammation but marked strawberry appearance

Mucosa	Fibrosa
16.30 per cent	0.10 per cent
41.80 per cent	0.17 per cent

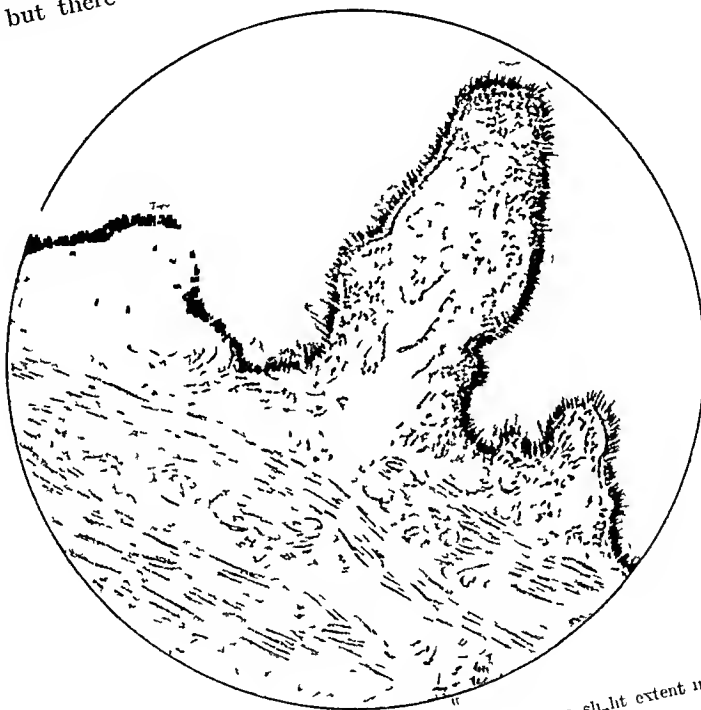


Fig 280—Lipoid at the base of the epithelial cell and to a slight extent in the stroma

thirds, including the fundus, was markedly thickened, the ridges were low, thick, and quite pathological, an inflammatory process had evidently been in progress for a considerable time, but not a trace of lipoid could be seen under the low power microscope. This suggests very strongly that the deposit of lipoid is an early phenomenon in the disease process, and that it may disappear—at least to the naked eye—at a later date.

Villi—In microscopic sections the lipoid is invariably most abundant in the villi—that is to say, the ridges seen on cross-section. It may be present in the epithelium of the surface, in the stroma of the villi or in both. The epithelium may be loaded, but more frequently a narrow line, stained bright red in Schriach R preparations, is seen running along the base of the cells the entire deposit being proximal to the nucleus (Fig 280). The deposit is more marked in the epithelium covering the villi than in that lining the intervening depressions. Indeed, one sometimes gets the impression that the villi are projecting upwards in order to collect or to absorb as much cholesterol as possible.

The *stroma* of the villi, that is to say the substance of the mucous membrane (what has been called the submucosa by some writers), although consisting mainly of loose connective tissue, always contains mononuclear cells, which are present in great numbers in conditions of inflammation (Fig 281). The lipoid is usually contained within these cells, imparting to them a granular appearance. In other cases it is completely extracellular. Frequently the two forms are combined. As a rule, when there is lipoid in the stroma it is also present in the surface epithelium, but in occasional specimens it was confined entirely to the stroma. Sometimes a trail of lipoid could be traced from the epithelium into the depths of the stroma, and down to the base of the villi. It almost appeared as if a snail had crawled down the villus, leaving a track of lipoid behind it.

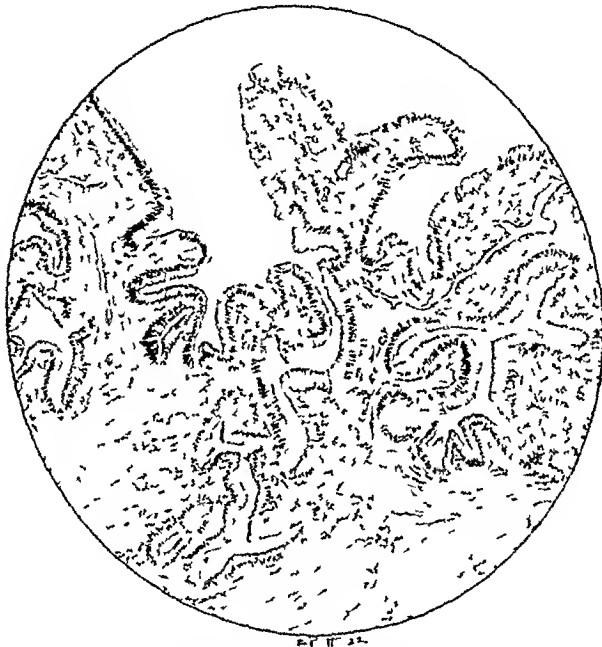


FIG 281—Gland like formation often seen in chronic cholecystitis. Infiltration with round cells is also well shown.

In some cases the *endothelium* of the blood-vessels contained streaks of material which stained red with Schiaraeh R. This interesting observation suggested the possibility that the lipoid might be absorbed into the blood-vessels. Careful search was therefore made for the presence of droplets of lipoid actually in the lumen of the vessel. These were never observed in the capillaries of the villi, but in two cases they were found in the vessels of the muscular coat. Too much stress must not be laid upon this remarkable finding, for the lipoid was only seen in Schiaraeh specimens, and as the tissue has to be treated for a few moments with alcohol, it is possible that some lipoid may have been transported so as to lie over the lumen of the vessel. The only convincing proof would be to demonstrate the lipoid within the lumen by means of the polarizing microscope, but owing to the difficulty of orienting the specimen when viewed by polarized light this is hardly possible. In one of the cases the lumen of the vessel appeared to be distinctly distended by the lipoid globulin, so the evidence, although not conclusive, is nevertheless suggestive.

The Fibrous Coat—Although the lipoid is always most marked in the mucosa, and is frequently confined to that layer it may also be found in the fibromuscular coat. Again it may be either intri- or extracellular. It is contained within cells which are of chronic inflammatory origin. Some of these are rounded or more often polygonal in form.

(Fig 282) Lipoid is best seen in this region where, as the result of long continued inflammation, the wall of the gall-bladder has become converted into a mass of organizing granulation tissue containing large numbers of young fibroblasts. These cells are often loaded with lipoid material. The occurrence of lipoid in the endothelium of small blood-vessels has already been referred to.

The extracellular lipoid may take the form of small granular masses, but not infrequently it is seen as narrow linear streaks, which may run at any angle to the surface. The appearance suggests that the lipoid is confined within tissue spaces or lymphatics, much as are the lines of epithelial cells spreading by a process of permeation from a carcinoma of the breast.



FIG. 282.—Inflammatory cells packed with lipoid granules. High power.

Lipoid occurs in the depths of the gall bladder under circumstances somewhat different from those which accompany its appearance in the mucosa. In the former case the wall is always the seat of advanced inflammatory changes, in the latter there may be so little change that the mucosa might otherwise pass for normal. In other words, the occurrence of lipoid in the mucosa is an early change, whilst in the fibrosa it appears at a much later stage.

To summarize the observations on the distribution of the lipoid. It may be found in any of the coats and at any depth. Its most common place of occurrence is the surface epithelium and the stroma of the villi. It is seldom found deep to the mucosa unless within chronic inflammatory cells.

V WHAT IS THE CAUSE OF THE LIPOID DEPOSIT?

Now that the nature of the lipoid has been determined, and its distribution in the gall-bladder well discussed, it is a pertinent question to ask what causes it to be deposited. A final answer to that question cannot be given at the present time. Experiments are in progress the object of which is to produce the lipoid deposits in animals under known conditions. But until success attends these efforts the answer can only be surmised.

The most probable factor is inflammation. In a study of the 52 cases in which lipoid was present inflammatory changes were found in every case. But the inflammatory changes were seldom marked, and were sometimes so slight in degree that it was difficult to be sure whether or not they were present. The changes were sometimes confined to the mucosa: at other times they extended into the deeper coats. The principal feature was a collection of the cells associated with chronic inflammation, namely, lymphocytes and plasma cells. Occasionally no inflammatory cells could be discovered, but presence of vascular dilatation and extensive œdema in the loose stroma of the villi indicated the action of some irritant.

When the inflammation becomes more severe in character, and especially when it reaches the stage of suppuration, the lipoid is no longer found. Can it be possible that, after being deposited, it has subsequently disappeared? One case already referred to suggests this possibility. The greater part of the gall-bladder was thickened and had evidently been the seat of inflammation for a considerable time. It showed no trace of lipoid. The remaining portion was much thinner, the mucosa was much less altered, but there were extensive deposits of lipoid. Such a case suggests that the early stage of inflammation, or it may be the milder forms of that process, are characterized by the deposition of lipoid, but as the inflammatory process progresses the lipoid disappears owing to some mechanism at which we can hardly even guess. The kidney offers some sort of analogy. In the early stages of nephritis the epithelial cells show an abundant deposit of fat, whereas in the stage of chronic interstitial nephritis this may have completely disappeared. The analogy, of course, will not bear too close a scrutiny.

It is somewhat different in the case of the deeper coats. Long-standing inflammation of the fibrosa, as evidenced by the presence of great numbers of fibroblasts, may be accompanied by extensive lipoid deposits within the inflammatory cells.

It must be admitted that the demonstration that the deposit of lipoid is associated with a certain degree of inflammation is in reality no explanation. We are still completely in the dark as to why the lipoid should be deposited. In a later part of this paper the relation of the gall-bladder to the cycle of cholesterol in the body will be considered. At this stage it may be suggested that if cholesterol should chance to be absorbed from the bile and to pass into the wall of the gall-bladder, any inflammatory or other process which interferes with that absorption may result in the cholesterol being deposited, first in the surface epithelium and later in the deeper parts either in the form of free cholesterol or of an ester formed by the union of cholesterol with a fatty acid. Should the villi be specially concerned in the process of absorption then the deposits would be most pronounced in those structures.

These are but guesses at the truth. Not until the deposition of lipoid has been produced experimentally in an animal will it be possible to state with certainty the factors which govern the formation of these deposits.

VI RELATION OF LIPOID TO GALL-STONE FORMATION

The etiology of cholelithiasis is a subject regarding which great uncertainty still prevails. A gall stone is composed of the several constituents of the bile combined in varying proportions. One or more of these may be absent. Indeed, only one may be present as in the pure cholesterol stone, the cholesterol-free stone found in hæmolytic jaundice etc. In view of these facts it is but natural that the attention of investigators should have been fixed entirely on bile in the attempt to determine the factors which govern the formation of calculi.

Three principal factors are held to be responsible for the production of gall-stones (1) *Stasis of the bile*, (2) *Infection of the bile with micro-organisms*, and (3) *An increase in the cholesterol content of the bile*.

1 **Stasis of the Bile**—This may occur as a result of a variety of conditions, such as obstruction interference with the innervation of the gall bladder muscular atony, etc.

2 **The Presence of Micro organisms in the Bile**—This is an important factor in determining the precipitation of substances usually held in solution. This has been

shown by many investigators When a specimen of bile is inoculated in vitro with a culture of *B. coli*, precipitation of the cholesterol and of the bile salts occurs This precipitation is probably due to interference with the bile salts, upon the presence of which the solubility both of the cholesterol and of the bile pigments depends

A clear distinction must, however, be drawn between infection of the bile and infection of the wall of the gall-bladder Just as it is frequently possible to find bacteria in the synovial membrane of a joint in cases of chronic synovitis when none can be detected in the synovial fluid, so bacteria may be present in the wall of the gall-bladder when none can be found in the bile Rosenow² has shown that in order to determine the existence of bacterial infection of the gall-bladder the wall of the bladder must be pounded up and added to the culture medium, the bile itself often being quite sterile Finkelstein, working in our laboratory, has demonstrated the same absence of bacteria from the bile in many cases of mild cholecystitis, strawberry gall-bladder, and biliary calculi When, therefore, we speak of infection as a factor in the production of gall stones we should think of the bladder wall rather than of the free bile

3 The Cholesterol Content of the Bile is doubtless an important factor in the production of calculi Two of the conditions most frequently associated with calculus formation, namely, typhoid fever and pregnancy, are characterized by a great increase in the cholesterol content of the blood and therefore of the bile But here again the danger in the past has been to overlook the importance of the gall-bladder itself As we have already shown, the wall of the gall-bladder may, under certain conditions, present an increase of the cholesterol content beside which any increase in the cholesterol in the bile fades into insignificance

It seems justifiable, therefore, to attempt to direct attention from the bile to the gall-bladder itself as the most important factor in calculus formation, at least in the early stages And it is the early stage which is all-important

A study of the material at our disposal leads one to the belief that the initial step in calculus formation is to be found in the wall of the gall-bladder itself, and consists in the deposition of a lipid in the form of cholesterol ester in the gall-bladder mucosa A glance at Fig 283 will show that as this deposit of lipid increases in bulk, the villus in which it



FIG 283.—Polypoid mass of cholesterol ready to separate. Early case of strawberry gall bladder. Stained with Schriach R

is contained may develop more and more into a papillomatous-like process, the stalk of which finally becomes so attenuated that separation is inevitable. When that occurs we have a foreign body composed of cholesterol and albuminous material, lying in the cavity of the gall-bladder, and forming an ideal nucleus for the formation of further deposits.

Whether or not this further formation will occur depends on a variety of circumstances. The factors which make for the formation of gall-stones are apparently periodic rather than continuous in their mode of action. In some cases, it is true, there may be variation, but shows a great variation in size and type. A collection of gall-stones from one case seldom size, as if formed at the same time. As a rule all the stones are of about the same this is usually variation of one whole set as compared with another set. For instance, in one of our cases there were three distinct sets of calculi in the gall-bladder, with about half a dozen stones in each set. The first were large, the second medium, and the third very small but all those of the same set were exactly the same size. The initial factor which started the stone formation must have come into play three times.

The two factors most liable to periodic fluctuation are the cholesterol content and infection. The cholesterol content of the blood and of the bile, for instance, will rise with each pregnancy, only to fall to normal in the intervals. Infection, again, is probably often periodic. The kidney in Bright's disease, the heart valves in endocarditis, may be the seat of recurring infections, with each of which the organ is left more and more damaged. So also with the gall-bladder. The profoundly altered gall-bladders which we often encounter are not as a rule the result of a single infection but represent the cumulative effect of many such attacks. The infection, however, is one which affects the gall-bladder wall rather than the bile.

From the above considerations it is apparent that there are several things to be said in favour of the view that the starting-point of a calculus may be the wall of the gall-bladder. It is interesting to note that in a book by Chauffard³ just published the author arrives at somewhat similar conclusion. Small biliary calculi were found to originate inside the villi as minute collections of cells surrounded by cholesterol. These are shed, grow, and ultimately become faceted. Dewey⁴ has succeeded in producing gall-stones in one rabbit by means of long continued injections of cholesterol. When these were embedded in paraffin by the method of Aoyama,⁵ sectioned, and the sections stained with methylene blue it was found that disintegrated cells formed the framework of these stones. Dewey considers that these represent desquamated cells, but it is more probable that they are epithelial cells of the surface in which the cholesterol was first deposited, and the cells subsequently shed.

It in no way follows that this is the only method of calculus formation. Indeed, when one looks at the calculi collected from a large series of cases, the variations are seen to be so extreme that one shrinks from being in any way dogmatic about the possibilities of their formation.

VII THE CLINICAL SIGNIFICANCE OF LIPOID DEPOSITS

The surgeon of course asks: What is the clinical significance of these deposits of lipoid in the gall bladder mucosa? What is the meaning of the strawberry gall-bladder, and how may it be recognized clinically? This is a difficult question to answer, for the problem is not a clear cut one. If the strawberry gall bladder formed a definite separate clinical entity it would be easy to go through the histories of such cases, and arrive at some fairly satisfactory conclusion. Unfortunately there are all degrees of lipoid deposit from no such disease as a strawberry appearance is most pronounced to those in which the lipoid is invisible to the naked eye, and finally there may or may not be present such a complication as calculi which may completely overshadow any symptoms which might be characteristic of the deposition of lipoid.

The only safe method is to select those few cases of typical strawberry gall bladder in which no complicating factors are present. A brief summary of three such cases may be given.

Case 1—Mrs W, age 30, weighing 160 lb, has suffered for ten months from what she described as pulling and drawing sensations in the epigastrium. She never had an acute attack of pain, nor was jaundice present at any stage of the illness. Nausea was a prominent symptom. After partaking of a few mouthfuls of food she feels as if she could eat no more. It seems to her that food is unable to pass the stomach. She does not complain of acid eructations or belching of gas. There is no periodicity to the sensation of abdominal discomfort, nor does food afford her any relief. Duodenal drainage by Dr Finkelstein showed numerous pus cells in the gall bladder bile, but no micro organisms. At operation the gall bladder appeared almost normal, apart from some slight thickening and opacity.

Further examination revealed an extreme condition of strawberry gall bladder. There were enormous deposits of lipid in the form of doubly refractive calculi crystals, and giving a marked reaction with the sulphuric acid test, throughout the whole of the mucosa. There were slight inflammatory changes both in the mucosa and the fibrous coat, but it is difficult to account for the presence of pus cells in the bile obtained by the duodenal tube.

Case 2—Mrs S, age 43, weighing 165 lb, complained of pain in the epigastrium and the right side of the abdomen. The present attack commenced shortly after labour three weeks ago. During the last few months she has had two similar attacks. The pain is usually localized to the right hypochondrium, but is sometimes referred posteriorly to the area between the scapulae. Food appears to stick in the epigastrium. For about an hour after a meal she belches gas. She feels better when hungry. The type of food and the size of the meal appear to make no difference. The gall bladder when removed was very slightly thickened and contained no stones. Only small patches of lipid could be seen here and there on the surface of the mucosa. Sections, however, showed an abundant deposit of lipid in the walls of the blood vessels. The mucosa was crowded with inflammatory cells, and the low power binocular microscope showed the ridges to be so swollen as to remind one of the convolutions of the brain.

Case 3—Mrs S, age 50, weighing 190 lb, has suffered from severe digestive disturbance over a period of two years. She has never had any violent attacks of pain such as might suggest the passage of a calculus, but the pain has been sufficiently distressing in character, and there is marked tenderness over the gall bladder. She has never been jaundiced. At operation the gall bladder appeared so normal that the surgeon hesitated some time before removing it. When the gall bladder was opened the entire inner surface was seen to be studded with the yellow specks characteristic of lipid deposits. The summits of the ridges were thickened and loaded with lipid, but the sides were thin and apparently free. No stones were found, but there was one large, yellow, papillomatous process. Sections showed enormous deposits of lipid in the stroma of the villi, little in the epithelium, and a considerable quantity in the fibrous coat. No inflammatory cells were observed, but the small vessels of the mucosa were congested, and an extensive oedema of the villi formed a striking feature of the microscopic picture.

Summing up this part of the subject, these cases showed fairly characteristic symptoms of cholecystitis without calculus formation, they were all distinguished by marked deposits of cholesterol ester, although without the formation of stones. The question as to why these pathological changes in the gall-bladder should produce, or at least be associated with, these gastric and other symptoms is one which the present writer does not feel himself qualified to discuss. In any case the explanation of the mode of production of symptoms in gall-bladder disease is often a matter of extreme difficulty. On this day of writing I was present at an operation for removal of the uterus because of fibroids, sweeping his hand round the abdomen the surgeon discovered that the gall-bladder was literally packed with stones, and much fibrosed as the result of long standing inflammation, and yet this patient had never had even a suggestion of symptoms pointing to disease of the gall-bladder.

VIII THE COMPARATIVE ANATOMY OF THE GALL BLADDER

One of the most curious facts about the gall-bladder, and one which presents itself as a stumbling-block in the way of any explanation of its function, is the absence of the organ in a number of animals. Amongst the animals mentioned by McMaster as having no gall-bladder are the horse, the deer, the rat, the pocket gopher, the dove, and the peccary. Why the mouse should possess a gall-bladder, but not the rat, the pocket gopher, but not the striped gopher, is certainly a knotty problem to solve. Our investigations on the gall-bladders of animals commenced in an endeavour to determine the histology of the normal gall-bladder. As already mentioned, autopsy

material proved quite unsuitable, and the surgeons were not presenting us with normal gall-bladders

Sections were accordingly cut of the fresh gall-bladder of a dog, they were cut on the freezing microtome and stained with Sehriach R, simply because that was the routine method we employed. Naturally we expected to find a normal mucosa without a trace of lipid. Picture our surprise when, on looking down the microscope, we found that the surface epithelium was packed with lipid which stained a brilliant red. The question at once arose. Was the presence of lipid in the gall-bladder of the dog a normal occurrence, or was it a manifestation of disease? In order to answer this question a series of dogs was examined, and observations were made on a number of other animals.

The Dog—Fifteen dogs were examined. In no case did the gall-bladder show any evidence of inflammation. In every case lipid was present in the mucosa, although varying in amount in different animals. It was always confined to the epithelium, and was never observed in the stroma. Moreover, the distribution in the epithelium differed from that of the lipid of the strawberry gall bladder in man. It invariably occupied the distal part of the cell so that the nucleus was closely pressed against the base of the cell. In man, on the other hand the lipid was usually proximal to the nucleus. The largest deposits were in the tips of the villi, and the deposit was often scanty or absent in the depressions between the ridges. In two cases the reaction with Nile blue sulphate is worthy of note. Instead of the dark blue or violet colour characteristic of cholesterol ester the lipid took on a rose pink somewhat resembling that of the neutral fat in the subserous coat.

The behaviour of the lipid under crossed Nicol's prisms was peculiar and perplexing. In some cases it appeared, as might be expected, in the form of brilliant white masses. Moreover, bright Maltese crosses were frequently seen adhering, so it seemed, to the surface of the villi. In the other cases, however, the material, which had stained so brilliantly with Sehriach, now appeared merely as a dull grey, and sometimes not at all. It really seemed as if the lipid varied in its physical characteristics in the different cases. With the closed diaphragm it had a dull grey colour. No evidence of yellow lipid could be seen either with the naked eye or by means of the low-power binocular microscope.

At one stage of the investigation it was thought that the lipid might bear some relation to diet, for it seemed to be more abundant in those animals which were poorly nourished. The matter was put to the test of experiment. A small piece of the gall-bladder was removed from a normal dog of average nutrition. The animal was then given nothing but water for seven days. At the end of that time another piece of the gall-bladder was removed. The dog was now placed on a full diet for a week, when it was killed, and the gall-bladder removed. The specimens were stained for lipid, but exactly the same amount was found in all three. Diet, therefore, appeared to have no effect on the lipid content of the mucosa.

In two cases masses of lymphoid tissue were encountered in the mucosa. These were similar in form and general appearance to the lymphoid follicles of the appendix. It was noticed that the epithelium covering these nodules was free from lipid. Similar structures are never found in the human gall-bladder.

In one dog used in experiments on the absorption of iron to be described presently, a remarkable appearance was observed. The gall bladder was removed after it had been injected with a 2 per cent solution of potassium ferrocyanide. The mucosa was found to be studded with small green nodules, which resembled green peas under the dissecting microscope. The contents were fluid or slightly gelatinous. Microscopic sections showed that the little bodies were vesicles or cysts in the substance of the mucosa. They were not found in any of the other dogs in which potassium ferrocyanide was injected into the gall-bladder.

The Cat—The gall bladders of five cats were examined. Lipid was present in two, absent in three. When present it presented quite a different picture from that seen in the dog. It was scanty in amount, took the form of minute droplets, and was confined to that part of the epithelial cell proximal to the nucleus. In the dog it was always distal to the nucleus. It was only with great difficulty that it could be seen with the

polarizing microscope, but several Maltese crosses were observed adhering to the surface of the villi.

Specimens of the kidney, the liver, and the adrenal were also examined for fat. The adrenal cortex, as might be expected, was loaded with cholesterol ester. Both the liver and the convoluted tubules of the kidney contained a large amount of material which stained red with Scharlach R. In the liver this material was brilliantly doubly refractive, but in the kidney not at all. This suggests that in the kidney the red staining material was a glycerin fat, in the liver a cholesterol fat.

Other Animals—The gall-bladder of the cow, the rabbit, the guinea-pig, and the frog were examined. In none of these was any lipid found.

IX THE FUNCTION OF THE GALL-BLADDER

In the history of medicine it has happened not infrequently that pathological investigations have thrown valuable light on the physiology of an organ. The case of the pituitary body and the localization of cerebral function are examples which at once suggest themselves. The same may prove true for the gall-bladder.

The function of the gall-bladder forms at present a favourite subject for discussion in surgical journals. We do not propose to review, as is often done, all the suggestions which have been put forward at various times. The gall-bladder is certainly not a mere reservoir for the bile, for its capacity is little more than one ounce, and it is probable that the daily production of bile amounts to nearly a litre. It has been suggested that a prime function of the gall-bladder is to convert the continuous flow from the liver into an intermittent flow into the duodenum. This view is strengthened by a consideration of the fact that the lower end of the bile duct is guarded by a sphincter (the muscle of Oddi) which relaxes in response to the demands of digestion, so that a considerable volume of bile can flow into the duodenum at the very moment when it is most needed.

This simple view entirely fails to explain certain facts which cannot be ignored. The recent experimental work of Rous and McMaster^{6, 7} has shown that the gall-bladder exerts a most remarkable concentrating effect on the bile which passes through it. In some instances the bile was concentrated as much as ten times. A simple reservoir does not concentrate the fluid which is contained within it.

Moreover, a study of the structure of the gall-bladder at once disposes of the idea that it is intended to play merely a passive role, such as that of the urinary bladder. A study of the structure of a part will often enable one to make a shrewd guess at the correlative function. Thus the glands of the stomach suggest secretion, the glands and villi of the small intestine suggest both secretion and absorption, the glands of the large intestine lined by cells containing mucin suggest the secretion of mucus. But the best idea of the essential features of the wall of the gall-bladder is to be obtained, not from microscopic sections, but from a direct view by means of the binocular dissecting microscope. The picture revealed by that instrument has already been described in some detail; it differs absolutely from the flat, featureless wall of the urinary bladder. The tall, graceful, delicate folds, thin as gossamer, covered by a highly specialized columnar epithelium, and plentifully supplied with blood-vessels, are surely designed for one purpose, namely, absorption.

Microscopic sections tend to confirm this view. The delicate villi with thin walled vessels running down the centre can play no part in a reservoir. They are constructed for absorption. The morphological evidence, however, shows that something is contributed by the gall-bladder to the bile. In a number of cases of mild inflammation the epithelial cells of the surface have been so distended with mucus as to become veritable goblet cells. A subsidiary function of the gall-bladder therefore appears to be the production of mucus, a function which, as might be expected, is called into increased activity in conditions of catarrhal inflammation.

It is sometimes stated, even in well-known monographs dealing with the gall bladder, that the mucosa is studded with glands. This is not the case. In chronic cholecystitis

however, the depressions between the villi become deepened and tortuous until their connection with the surface may appear to be cut off, so that they may be mistaken for glands, an error made all the more possible by the occasional distention of the cells with mucus.

In order to confirm or disprove the idea suggested by anatomical considerations a series of experiments was undertaken with the object of determining, first, whether absorption really does occur, and second, what is the constituent of the bile which is absorbed.

Absorption of Iron—The method employed for solving the first problem was the demonstration of the Prussian blue reaction in the wall of the gall-bladder after the injection of an iron salt into the lumen. The abdomen of a dog was opened, a fine needle introduced into the gall-bladder, the bile withdrawn, and an equal quantity of a 2 per cent solution of iron ammonium citrate injected. A series of dogs was used, and at varying intervals of time after the injection the gall-bladder was removed. In some cases the animal was kept under the anæsthetic, in others the abdomen was closed, and the animal allowed to live for twenty-four hours.

As soon as the gall-bladder was removed it was opened, the surface washed free of bile and the iron salt, and the specimen placed in a fixative to stop any diffusion of the iron. Pure formalin, 10 per cent formalin, and 95 per cent alcohol were used. The best results were obtained with pure formalin. The fixative contained in addition a 2 per cent solution of potassium ferrocyanide and 1 per cent hydrochloric acid. If absorption of the iron had taken place the Prussian blue reaction, as evidenced by the appearance of blue granules, would be observed within the wall of the gall-bladder.

The earlier experiments were inconclusive, partly owing to the diffusion of the iron throughout the wall, partly to the formation of blue deposits of mucoid material on the surface. When, however, pure formalin was used as a fixative, and when the surface of the mucosa was washed thoroughly clean before the fixative was used the results were clear and decisive. Even after so short a time as half an hour there were numerous blue granules in the epithelial cells, and to a lesser extent in the stroma of the villi. None were seen in the deeper layers, nor was there any indication as to whether the iron was absorbed into the blood-vessels or lymphatics.

A recent observation by Harer, Hargis, and Van Meter throws light upon this question. These workers introduced a hypertonic solution of potassium sulphocyanide into the gall-bladder of a dog through a ureteral catheter passed up through the ampulla of Vater. Lymph was collected in capillary tubes from the lymph channels in the wall of the gall bladder, and tested with ferric chloride. Positive results were obtained within a very short time after injection. This experiment suggests not only that the gall-bladder possesses ready powers of absorption, but also that the absorbed material passes into the lymphatics.

Absorption of Cholesterol—Granted that absorption may occur from the gall-bladder the question arises: What is absorbed?

From the work of Rous and McMaster² we have every reason to believe that water is absorbed. These investigations have shown that the bile becomes concentrated to a remarkable degree after a brief sojourn in the gall bladder. So great is the absorptive power of the mucosa that this concentration can be shown when the bile is allowed merely to flow through the gall-bladder without being kept there. Of the solid constituents of the bile (bile salts, bile pigments, cholesterol, and lime), the substance with which we are specially concerned is cholesterol. Is there any evidence that cholesterol is absorbed by the gall bladder?

Before this question can be answered it is necessary to consider briefly the part played by cholesterol in the animal economy. Here again we are more familiar with the pathological manifestations of the subject than with the behaviour of cholesterol in health. Deposits of cholesterol are found in arteriosclerotic blood vessels, in the white spots of albuminuric retinitis in anthrax, and in the kidney tubules in cases of nephrosis. The blood cholesterol is raised in pregnancy, convalescence after typhoid fever, chronic

nephritis, nephrosis, diabetes, jaundice, and many cases of cholelithiasis. It is lowered in acute infections (with the notable exception of typhoid) and in tuberculosis.

Cholesterol, isolated from gall-stones by Conradi in 1775, and fully examined as to its chemical constitution in 1815 by Chevreul, who first gave it its name of *cholesterin* (now usually changed to cholesterol to indicate its alcohol-like character), is a monatomic alcohol which on account of its solubility in fat solvents is regarded as a lipid. It is widely distributed in the animal and vegetable kingdoms, occurring in the latter in the form of an isomer named phytosterol. It is found in abundance in blood, bile, sebum, the white matter of the brain, the medullary sheath of nerves, the cortex of the adrenal, and the corpus luteum of the ovary.

As already indicated, our information regarding the physiology of cholesterol has not kept pace with the advances in our knowledge of its behaviour under pathological conditions. The chief contributions have been made by a single set of English workers, Doree, Ellis, Fraser, and Gardner,⁹ in a series of papers published in the Proceedings of the Royal Society between 1908 and the present year. Their work has consisted in feeding animals with cholesterol and estimating the variations in the blood cholesterol and the amount of cholesterol excreted in the feces.

The cholesterol in the feces should theoretically be derived partly from the food, partly from the bile. Doree and his associates found that the cholesterol in the feces of the dog amounted to only one-fifth of the total which might be expected from these sources. Moreover, the cholesterol in the feces could be entirely accounted for by the cholesterol in the food. That in the bile had apparently been absorbed, that it should have been destroyed is unlikely, owing to the very stable nature of cholesterol. The cholesterol of the bile, therefore, is absorbed, excreted, and reabsorbed once more. The truth of this cholesterol cycle is recognized in the title of a recent book by Grigaut.¹⁰

The question is, where does this absorption occur? It is here suggested that some, at least, is absorbed by the gall-bladder. The evidence in support of this view is by no means conclusive, but some experimental work, at present only in the initial stage, will, it is hoped, throw light on the subject. The following evidence is also worthy of consideration.

1 Under certain pathological conditions cholesterol is deposited in the wall of the gall-bladder. But is this cholesterol on its way in or on its way out? Is it being absorbed or is it being excreted? This question can hardly be answered on purely morphological grounds. The anatomical arrangements already discussed, however, suggest absorption rather than secretion. The occurrence of cholesterol in the connective-tissue cells of the mucosa, and possibly in the lymphatics, hardly suggests a process of excretion. Nor was of the opinion that all the cholesterol in the bile was produced by the gall-bladder. In this he was undoubtedly wrong. Adamu considers that, as usual, the truth lies midway between the two extremes, and that part of the cholesterol is produced by the liver, part by the gall-bladder. It must be admitted that the fact that cholesterol which may be passing inwards through the wall of the gall-bladder is deposited in conditions of disease does not constitute a proof that such absorption occurs under normal conditions, but it points in that direction.

2 The injection experiments of Dewey are also of interest. This observer found that, as the result of long continued injections of cholesterol, deposits of that substance were found in the liver and kidney of the rabbit. In some cases large deposits of a material which, although not anisotropic, yet stained red with Sudan III and purplish blue with Nile blue sulphate, were found in the mucosa of the gall-bladder. This substance which occupied the centre of the villus, he considers to be a cholesterol compound without anisotropic properties. He is of opinion that the material was situated within the lymphatics. If this is correct the cholesterol must almost certainly have been absorbed from the bile.

3 A consideration of the difference in concentration between liver bile and gall-bladder bile still further supports the view that cholesterol is specially selected for absorption. The bile in the cystic duct is very much more concentrated than that in the hepatic

duct, but the concentration does not affect all the solids alike. According to the figures given by Starling, sodium taurocholate is concentrated 16 times, sodium glycocholate 20 times, but cholesterol only 10 times. It would appear, therefore, that in addition to the absorption of water there is also some absorption of cholesterol.

4. It is only by means of experimental methods that any certain conclusion can be reached regarding this matter. The following preliminary experiment is interesting and suggestive, but this branch of the work is still at so early a stage that it would be unjustifiable to attach to it any undue importance.

The average blood cholesterol in a series of healthy rabbits was determined and found to be 0.25 mgrm. in 1 c.c. of blood. The variation from this figure was very slight, not more than 0.02 on either side. Five rabbits were fed on 0.1 gm. cholesterol daily. At the end of four days the gall-bladders of two rabbits (Nos. 4 and 5) were removed. The feeding was continued. At the end of the ninth day the blood cholesterol of all the rabbits was estimated. The results are given in Table III. In the animals with a gall-bladder the average was 0.333 mgrm., in those without a gall-bladder it was 0.215 mgrm. Something must have interfered with the absorption of the cholesterol. Many possible sources of error will have to be checked, such as the effect of the anæsthetic, of the laparotomy, etc., but for the present the most obvious factor is the absence of the gall-bladder.

Table III.—ILLUSTRATING THE EFFECT OF CHOLESTEROL FEEDING AND CHOLECYSTECTOMY ON BLOOD CHOLESTEROL IN THE RABBIT

Control (average)	25 milligrams per 100 c.c.
1 Fed cholesterol	38
2 " "	30
3 " "	32
4 Cholecystectomy	21
5 " "	22

The cholesterol in the blood is derived partly from the food, partly from the bile. The work of Doree and his associates has shown that the cholesterol in the fæces accounts for that in the food, so that the cholesterol in the bile must be absorbed. We have seen that the architecture of the gall-bladder is beautifully designed for purposes of absorption. We have seen that the gall bladder is capable of absorbing solids as well as water. We have seen also that cholesterol is deposited in the wall of the gall-bladder in pathological conditions. The absorption of cholesterol is evidently interfered with in some way in animals from which the gall-bladder has been removed. It is suggested that the main avenue of this absorption of bile cholesterol may be the gall-bladder, and that possibly this may constitute an important function of the gall-bladder.

In these days when the gall-bladder is regarded with such dark suspicion, and is offered up, an innocent victim, on many a surgical altar, it may be well to recall that possibly after all this much condemned organ may serve some useful purpose in the animal economy.

SUMMARY

1. A study of the gall-bladder with the binocular dissecting microscope reveals a new view of the anatomy of the organ, and throws suggestive light on the question of its function.

2. That function is undoubtedly one of absorption, and it is possible that one of the chief substances absorbed is the cholesterol of the bile.

3. The formation of deposits of cholesterol ester in the mucosa of the gall-bladder is an important feature in many cases of early cholecystitis.

4. These deposits occur both in the surface epithelium, in the connective-tissue stroma and possibly in the lymphatics.

5. In some cases at least the first step in the development of gall stones may consist in this formation of cholesterol deposits.

6. A new microchemical test for cholesterol in the tissues is described.

7 The normal gall-bladder of the dog contains a peculiar lipid material in large amount. That of the cat contains a much smaller amount of the same material. In none of the other animals examined was it found.

In conclusion, I wish to express my indebtedness to those who have made this research possible—to the surgeons of the Winnipeg General Hospital for willing assistance in studying the clinical side of the subject, to Professor A. T. Cameron of the Department of Biochemistry of the University of Manitoba for much invaluable help with the chemical work and, amongst other helpers, to Miss Olive Lightcap and Miss M. van Romburgh for their technical assistance.

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X-RAY PRINTS A SUGGESTION.

By A P BERTWISTLE, LEEDS

It had always struck me that much of the value of x ray prints in text-books was lost owing to absence of contour of the soft parts. I have accordingly tried to define the outline of these parts in such a way as to render the pictures more real



FIG 284

I began by outlining the edges of the parts with a paste of bismuth and paraffi. This was unsatisfactory, owing to the difficulty in marking the exact margin



FIG 285

I then found that, whilst skin shows quite clearly on the negative, it does not do so on the print, though occasionally the apparent soft parts are represented by structures

deep to the deep fascia I am given to understand this is due to the printing being done solely for bone definition I accordingly took a series of negatives, and with a point outlined the skin The background was subsequently filled in with Indian ink, giving the effects reproduced in the accompanying prints



FIG 286



FIG 287

I would suggest that text-book illustrations would have a greater value if done by a similar method, which should be equally valuable for orthopaedic work

The illustrations shown (Figs 284-287) are used, together with other similar ones, in the casualty reception room for instructional purposes

VISITS TO SURGICAL CLINICS AT HOME
AND ABROADTHE MAYO AND CRILE CLINICS WITH SPECIAL REFERENCE
TO THYROID SURGERY.

BY W H BOWEN, CAMBRIDGE

ROUND the great lakes of America thyroid disease of all sorts is endemic, and the abundance of clinical material with its pathological harvest makes the study of thyroid disease intensive and authoritative. The present paper is based on the work seen at Dr Crile's clinic at Cleveland and the Mayo clinic at Rochester, and upon the opportunities given for discussing cases and points, courtesies so freely extended to the visitor, and which helped to enhance the value of a visit to these celebrated clinics. In the Mayo clinic I heard Dr Boothby and Dr Plummer lecture on thyroid disease, and was shown over the Basal Metabolism Department by the former, every facility being afforded for observation, coupled with invaluable verbal explanations. I also heard much discussion on the differential diagnosis between adenoma thyroid with hyperthyroidism, and exophthalmic goitre, a subject which the Mayo clinic has put on a firm basis by means of its wide experience and careful analysis of its cases. There is a certain friendly rivalry between the Cleveland and Rochester clinics which is of some advantage to the visitor, since the fact that he has been to the one affords ample opportunity of discussion in the other. Both these clinics are controlled by men of great skill and big outlooks, so that discussion cannot but broaden the view and enlighten the mind, and criticism in either school calls forth the very best in the way of clinical or experimental study for the student who seeks knowledge, and it is from such opportunities and without any reference to book work that I write this paper. As a matter of fact, I carefully avoided furnished my American literature on the subject of thyroid disease until this paper was ordered to gain some idea of the opinions of our leading authorities on the subject of thyroid surgery especially and to get some impression as to the comparative standard of achievement.

One ought not to overlook a great advantage which American surgery possesses in the centralized clinic system which tends to the free communication and interchange of opinions between the departments, especially between the pathologists, internists, and surgeons. They all have their rooms and do their work in the same central building—the clinic building when a problem is hot in their minds whose opinion is most valuable and who can supply the special knowledge required either to clear up the doubt or to suggest those lines of inquiry most likely to prove beneficial. This is real team-working, and is probably one of the greatest assets of the American clinic system, for it has reached a level of organization which is I think unknown in this country. I shall have more to say on this matter when writing on Crile's surgical work on goitre—the finest demonstration of the perfection of a team system which I have ever witnessed, or, I believe, am ever likely to witness.

The main interest of this paper may be epitomized as follows: first will be given the Mayo clinic classification of goitres, secondly some reference to the Basal Metabolism Department of the same clinic, thirdly the mode of operating, with special reference to Dr Crile's clinic, and finally, a few remarks on thyrotoxicosis.

The Mayo clinic classification of thyroid disease given by Dr Boothby is as follows:—

- 1 Colloidal diffuse enlargement, which is identical with what, in this country, we call simple parenchymatous enlargement
- 2 Adenoma (a) without hyperthyroidism
- 3 True exophthalmic goitre
- 4 Thyroiditis simple and tuberculous
- 5 Malignant thyroid

This classification is a good working basis for the purpose of this paper, which in part aims at emphasizing the way in which differential diagnosis has been forwarded in America. The Mayo clinic attaches the greatest importance to the differential diagnosis between adenoma thyroid with hyperthyroidism, and exophthalmic goitre. Interest centres round this problem, as it always will do around differential diagnosis, especially when the difference approaches some degree of refinement of diagnosis, and when there is clash of Olympic opinion, for this is the bone of contention between Crile's clinic and the Mayo clinic.

Before going any further let me briefly get rid of headings 1, 4, and 5. Colloidal diffuse (parenchymatous) goitre may be dismissed with the brief reminder, well known to all, that operation is only required for æsthetic or mechanical reasons. When associated with nervous symptoms, however, Dr Plummer contends that it may lead clinicians astray. This will be referred to later. Thyroiditis may be dismissed as being devoid of special interest, on account of its rarity and its special clinical characters. Malignant thyroid again has special characters and features of its own which put it in a grouping by itself and on the whole render its diagnosis one of no great difficulty.

Before leaving the subject of classification, let me say that the Mayo clinic realizes that a classification is but an effort to produce order out of confusion and provide some sort of basis for discussion, and that it is not final and unchangeable. Dr Boothby told me, after giving this classification, that it was an interesting fact that in the last three years, among some two thousand goitre cases, they had four cases of diffuse colloidal enlargement of the thyroid with all the symptoms of exophthalmic goitre, and which they called exophthalmic goitre. Also, occasionally a case of adenoma thyroid became a definite case of exophthalmic goitre. They gave no explanation of these anomalies, but stated them as facts which must be accepted, however inexplicable they may appear at present.

The Mayo clinic contends that there is a wide difference between adenoma thyroid with hyperthyroidism and exophthalmic goitre, that the outlook in the two classes of case is widely different both in operative risk and curative result. Furthermore it contends that others have confused one type of case with the other, have grouped them all together under the heading of exophthalmic goitre, with a resultant low mortality from operation which would not have been achieved had the differential diagnosis been more carefully considered.

To those outside the controversy this endeavour to differentiate between two conditions clinically very similar, and both apparently requiring the same treatment, may appear as a refinement in diagnosis which is uncalled for, but longer and more careful consideration of all the factors involved, and especially the difference in operation risks, will, I believe, decidedly alter any opinion based on a superficial survey of the subject, and I venture to think it is well worth while to go over the points in differential diagnosis which the Mayo clinic emphasizes.

Dr Boothby says that adenoma thyroid with hyperthyroidism and exophthalmic goitre are two diseases as separate as typhus and typhoid fevers. He considers adenoma thyroid with hyperthyroidism as the normal condition plus excess of thyroxin, but in exophthalmic goitre there is something more than excess of thyroxin. At the present

time he is not prepared to say wherein this difference lies, but mere speculation might suggest that in exophthalmic goitre there is some alteration in the thyroxin molecule.

The points in differential diagnosis which the Mayo clinic emphasizes in separating adenoma with hyperthyroidism from exophthalmic goitre may be roughly classified under four headings (1) *History*, (2) *Physical signs*, (3) *Symptoms*, and (4) *Difference in metabolic rate*.

1 An important point in the history of the case is the length of time the disease has existed. The time which a case of exophthalmic goitre takes to develop is on an average eight months, whereas in a case of adenoma with hyperthyroidism, the signs and symptoms take years to develop, being preceded by a stage of adenoma without hyperthyroidism, for the one usually merges into the other. Thus adenomata begin somewhere about the age of 20 years and exist for about 15 to 20 years before taking on toxic characters, the average over a number of cases being 17.7 years. It will be gathered from this that the onset of signs and symptoms is much more clear and definite in exophthalmic goitre than in adenoma with hyperthyroidism.

2 The physical signs calling for comment are the enlargement of the gland and the exophthalmos. As would be expected, the adenomatous gland is asymmetrical as compared with the true exophthalmic goitre, but also it is noted that the very large glands are the adenomatous ones. I am not prepared to comment upon the statements made to me, but would here remark that this apparent differentiation between the two types is of course relative, and, to those who are used to diagnosing tumours, distinctions which may appear obvious on paper sometimes provide great difficulties in practice.

Exophthalmos with adenoma is uncommon and may even be said to be rare, and when present is but slight compared with the staring eye seen in exophthalmic goitre. On the other hand, exophthalmos was present in 60 to 80 per cent of cases of exophthalmic goitre.

3 Under symptoms the following are the main points emphasized. Adenomatous cases do not get gastro intestinal crises, which are peculiar to true exophthalmic goitre, is also are thrills and bruits, the nervousness of exophthalmic goitre and that of hyperthyroidism are two very different things which experience soon differentiates, the appetite in exophthalmic goitre is large (unless there are gastric complications) whilst the weight is stationary or falling, as a consideration of the metabolic changes would lead one to expect.

4 The basal metabolism is never as high in adenoma with hyperthyroidism as in exophthalmic goitre.

Before giving some account of the method of estimating the metabolic rate and discussing its diagnostic and prognostic significance, I would like to refer to a lecture I heard given by Dr Plummer at the Mayo clinic, in which, leaving aside the differential diagnosis between adenoma thyroid with hyperthyroidism and exophthalmic goitre, in the elucidation of which he has played so great a part, he concentrated on an entirely different subject, namely the differential diagnosis between the colloidal diffuse goitre (simple parenchymatous) with psychoneurotic symptoms, and exophthalmic goitre. It is a less important group of cases than the adenomata as one would expect, and I cannot say whether it may not have been exaggerated by the war. I remember Dr Boothby mentioning this differentiation, but evidently as of comparatively small importance. Looking back, I am inclined to think that this is one of the refinements of diagnosis which are the outcome of the searching investigation and the complete report which the clinic writes on every case.

In distinguishing between these two groups of cases Dr Plummer's chief points were —

a The pulse. If the patient were only nervous he would be able to tell the physician that when his nervousness under observation makes his heart beat rapidly, yet there are times when he knows that it is slow or relatively slow.

b Cases of exophthalmic goitre have weakness of the quadriceps muscle noticeable when they are asked to step up. The value of this diagnostic point may be demonstrated

in this way. The exophthalmic goitre is not lacking in self-confidence, whereas usually the psychoneurotic is. If the exophthalmic case is asked to step up on to some high stool he will cheerfully acquiesce and fail to fulfil his intention, whereas the psychoneurotic can only with difficulty be persuaded to make the attempt, but steps up without the slightest trouble.

c There is increased appetite in the exophthalmic case, as previously mentioned. d Exophthalmic goitres are relatively intolerant of heat. The metabolic rate and increased intake of food show they are living at an excessive rate, with dilated surface vessels (flushing and sweating) as part of the body activity. This all means that they are burning to excess with the subjective sensation of heat.

e The metabolic rate is increased in exophthalmic goitre, as mentioned in (d). In summing up his lecture, Dr Plummer said that the three main points in differential diagnosis to which the clinic attached importance were: (1) Increased appetite, (2) Intolerance of heat, (3) Increased metabolic rate, all of which pointed to exophthalmic goitre. He stated that they had found no trouble in the differential diagnosis between exophthalmic goitre and early tubercle.

Basal metabolism, or the basal metabolic rate, is the method of estimating the metabolic changes occurring in the body during a certain period of time. It may be said to be the expression of the cellular activity of the body. At the Mayo Clinic the estimations are carried out on a very large scale. They attach importance to the results, but are very careful not to attempt to influence the surgeon unduly in deciding on his mode of treatment or the prognosis. Dr Boothby, who is in charge of the department, with the title of Chief of the Department of Clinical Metabolism in the Division of Medicine, compares the records obtained with those of a temperature chart in its bearing on the diagnosis and prognosis of a fever, which anyone will agree is a very modest position to take up, although entirely in keeping with his scientific attitude. He points out that he advises neither the line of treatment nor even the type of operation. He supplies certain data which, forming part of a whole, play a part in the making of a diagnosis and deciding the line of treatment.

The department is in a wing of one of the top floors of the Colonial Hospital. It comprises dressing-rooms for the patients and the rooms where the clinical investigations are carried out on one side of a corridor, and on the other a large laboratory, staffed by girls, where the gas analyses are made under supervision, and, adjoining this, private laboratories for Dr Boothby and his colleague, Miss Sandiford. The division given up to the clinical investigation of patients consists of two rooms alongside each other with a small ante-room common to both, where the expired air is collected into special gas chambers. The patient on arrival lies on a couch for about twenty minutes so as to enable him to be quiet and comfortable and free of undue physical or mental stimuli. A respirator is then put over his mouth and nose, with a mask so constructed that there shall be no escape of air at the side. This respirator has attached to it two thick rubber tubes, one connected with the outside air, the other going to the collecting chamber mentioned above, to do which it passes through the wall separating the room from the ante-chamber. The respirator being applied, the collecting of the expired air are carried out with a moment timed by three observers and activated by the turning of a tap.

The analyses of the oxygen apparatus, and from this the rate of tissue change is estimated. It appears that the estimation of the basal metabolic rate is essentially of value in thyroid disease, and its value as a clinical adjunct is practically limited to the study of this disease. It is the measure of the cell activity, and its great value is in deciding whether a patient has exophthalmic goitre, and the rate of tissue waste which is going on. Its bearing may be summed up in the words of Dr Boothby that in exophthalmic goitre the weight and the basal metabolism are the two important factors. If the rate is over 50 it means a severe condition, but with that statement came the reiteration of the warning that the Department of Clinical Metabolism does not decide what can or should be done in the way of operation.

At the Mayo clinic the Department of Basal Metabolism was opened in March, 1917, and by the end of that year 1143 estimations had been made. They have been carried out on much the same scale ever since, so that it will be realized that it is no longer an experimental inquiry. On the other hand, Dr Crile is sceptical as to its value, and although he has it carried out in his clinic it is entirely subsidiary to the clinical examination. At Cleveland it may be said to be still on trial, the precariousness of its reputation resting on the possible margin of error.

Before considering operative treatment, let me refer briefly to other modes of treating the diseases of the thyroid. It will be realized that the problem is essentially the treatment of exophthalmic goitre, for the other forms of enlargement sink into insignificance compared with the risks run in operating on the true exophthalmic goitre case.

X rays are abandoned. It has not been proved that they affect the course of the disease materially. Iodine is not advised in cases of adenoma, save in small doses and over a short period of time. Dr Marine says that its free use over an extended period is liable to set up hyperthyroidism. I heard Dr Charles Mayo say in the operating theatre that thyroid extract can be tried in young people, but in older patients it caused degenerative changes and was to be avoided. There is no other drug treatment save what is symptomatic, such as digitalis for cardiac embarrassments.

The treatment in Crile's clinic and the Mayo clinic of all forms of goitre except the colloidal diffuse (parenchymatous) is operative. Preferably a thyroidectomy is done. If this is inadvisable on clinical grounds, ligation of the superior thyroid arteries is performed. Both sides may be operated on at once, or after an interval of a few days. Dr Crile allows an interval of three days to elapse between the tying of the two vessels. After the ligation of the superior thyroid arteries the patient is sent home for two or three months. She then returns for further examination, and usually a thyroidectomy is done. The beneficial result of ligation, which is unquestionable, is differently explained by the two clinics. In Crile's clinic it is said to be due to the tying in of the nerves with the vessels, whilst the Mayo clinic insists that it is entirely a matter of partially cutting off the blood supply. In neither of these clinics did I see any attempt to ligate the inferior thyroid vessels. The injection of the gland with hot water is still occasionally done in the Mayo clinic, but Boothby said its chief value was in seeing how the patient reacted to any form of surgical interference.

In the Mayo clinic the goitre patient has no differentiation from others. The morning work usually begins with thyroid crises, but they are brought into an empty theatre and put straight on the operating table, for anaesthetic rooms are unknown. Ether by the open method is usually given, and when the anaesthesia is induced the surgeon and his assistants and visitors enter the theatre and the operation begins. In Crile's clinic, again, there are no anaesthetic rooms. Usually the anaesthetic is entirely a local one, $\frac{1}{2}$ per cent novocain supplemented by gas and oxygen if necessary, and occasionally just a little ether. At one time Dr Crile adopted the method, which is now well known, of taking the patients up to the theatre and bringing them back again without operating, in order to see how they reacted and to get them used to the novelty of going to an operating theatre. This he has abandoned, but now often operates in the patient's room, the patient lying in bed. He says they have proved that the carrying of the patient to the theatre has a definite risk of its own. This of course refers to cases of exophthalmic goitre of the more severe sort. Another preliminary measure employed is to try the patient with gas and oxygen beforehand. This is scarcely more than picking up an acquaintanceship with the anaesthetist and the gas and oxygen apparatus. Dr Crile says that in cases of exophthalmic goitre they have reduced the shock of operation to the point where the admission of the patient to hospital is a bigger shock than the operation itself.

Although thyroidectomy is the treatment of exophthalmic goitre, this does not mean that the condition is looked upon as primarily a disease of the thyroid gland. The view is that the thyroid element in the disease is comparable to a link in a chain. It is, as far as is known, it presents the only tangible link, and from the point of view of treatment, the only one open to direct attack. Hence thyroidectomy is openly recognized, not as a

specific treatment, but as a line of attack which, in the absence of any other form of treatment, justifies itself on theoretical grounds. It is now generally recognized that this theoretic principle is consolidated by the surgical results.

The operation of thyroidectomy in America is much more complete than any I have seen in this country or any I have seen described in British literature. The operation of resection is consolidated by the surgical results. The operation which is in any way comparable to the operation seen in America—Thyroidectomy is not an operation limited to Cleveland and Rochester, for I saw it carried out at Baltimore and Chicago. It consists in the removal of both lobes and the isthmus, save for a posterior portion of each lateral lobe equivalent to about one quarter to one-sixth of the total volume of a normal thyroid gland. It is not a hemithyroidectomy, but a final operation, and it shows no hesitancy in cutting across the gland substance in order to leave the extreme posterior part of each lateral lobe behind.

The advantages claimed for the operation are a removal of a maximal amount of diseased gland with a maximal benefit, the retention of sufficient glandular tissue to prevent myxœdema, and—by leaving the posterior surfaces of the lateral lobes—the risk of damage to the recurrent laryngeal nerves is reduced to a minimum, the parathyroids are not removed, and the lateral surfaces of the trachea are untouched. The incision and method of approaching the gland do not call for comment. That it is a very severe operation will be recognized by anyone who has operated much in this line of work. In the hands of the expert it has the appearance of simplicity of technique which is so noble to be the undoing of the novice. I saw many operations in the Mayo clinic and in Crile's clinic, both ligations and thyroidectomies, and never saw any trouble. I was seeing expert master surgeons operate, and skill and speed were combined.

Gentleness of manipulation as a cardinal rule of operating is coming into general recognition, but in a condition like exophthalmic goitre it ought to be looked upon in the same light as aseptic technique. I have never seen delicacy of touch, gentleness of handling, and speed of operating, carried to so high an artistic pitch as is seen in Dr Crile's work. The scheduled time for a thyroidectomy at the Lakeside Hospital is fifteen minutes, and it is done in the time. Not only done, but almost bloodlessly, and so methodically as to give the impression of simplicity even where a goitre of large size and great vascularity is concerned, and whilst speed is recognizable—for all the operator's movements are the quick, active movements of a virile intelligence—there is no hurry and no sign of strain or anxiety anywhere in the theatre. This is due not only to the skill of a master surgeon, but to the foresight and capacity for organization without which speed and accuracy in operating would not suffice.

Team work, an expression which has become so common on men's lips in this country in reference to certain crude efforts at organization, is seen carried out to plan in the greater American clinics. In both the Mayo and Crile clinics, when a surgeon comes to operate he always operates in the same theatre or group of theatres, and with the same assistants and accessories. Surgical skill combined with an almost perfect team system is what enables Dr Crile to complete a thyroidectomy in fifteen minutes, not in a special show case, but in a series of cases. Is the time likely to be improved upon? I doubt it. Dr Crile at the Lakeside Hospital at Cleveland has three theatres, five assistants, and a highly trained set of theatre nurses. His patients are operated on in the same gowns, towels, swabs, and gloves, whether they assist, the same nurses, the same theatre, with the same instruments, the same come from the almshouse round the corner or the home of the city hospital. The splendid American medical centres the nursing home is part of the institution called a nursing home in this country. At the Lakeside Hospital, as at the Boston hospital and those I saw in other cities, the nursing home is an annexe to the hospital. It is a part of the hospital built with single rooms instead of wards. But beyond this the gulf between the hospital and nursing home does not extend. That we shall have to follow the American system in this country, I have no doubt at all, and the surgeon of fifty years

hence will no doubt smile sympathetically when he reads of what his predecessors put up with in the early post-Listerian era. The change will have to come gradually, and I think will best be realized by building an annexe in the form of a separate block to an existing hospital, using the hospital theatres, pathological departments, and entire staff alike for the charity and paying patients, or, in the case of large centres, by building entirely separate paying hospitals, as has been done already in Birmingham and other large towns.

Team work is seen to perfection during a thyroidectomy at Cleveland. I have represented in the accompanying figure (Fig 288) the positions of the various members of the surgical team or service. The nurse anaesthetist is ready at the patient's head with a gas oxygen apparatus, but this is only given at the surgeon's orders. Infiltration anaesthesia with $\frac{1}{2}$ per cent novocain is the usual anaesthetic. If this local anaesthetic only is used the anaesthetist sits close to the patient's head and talks to her all the time very quietly and soothingly, or firmly, as may be necessary. Dr Crile stands at the patient's left side.

He makes the skin incision with the left hand—being ambidextrous—but after this he does most of the work with the right hand. Practically he cuts all the time. Serious traction is never seen. Occasionally he may put a finger in the wound to feel the limit of the growth and to separate gently where such can be done easily and expeditiously, but never otherwise. He has no respect for muscles if they obscure a view of the operation area. Should the thyroid go under the sternomastoid, he divides the muscle in order to expose fully the limits of the gland. One never sees a retractor used. He but rarely picks up a Spenceer Wells, all the picking up of vessels being done by his chief assistant, who stands opposite to him. The second assistant, who stands to the left side of the head

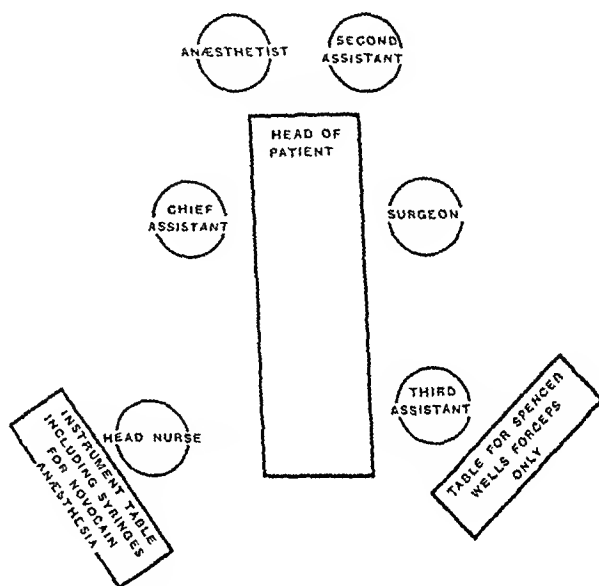


FIG 288—Diagram to show the position of the operating staff and the tables.

of the table, constantly, and yet without acting as an obstructionist, mops the wound dry. There is a third assistant who stands at the left of Dr Crile near to the bottom of the table, and his duty is to supply the chief assistant with Spenceer Wells' utility forceps. He has literally dozens of them, for the whole wound seems hidden by them when the operation is completed. The almost automatic character of this thoroughly organized assistance is seen in the way the first assistant puts his hand out towards the fluid without looking, and the Spenceer Wells is not only put into his hand but in such a position that it can be used at once. Whether as an exhibition of surgical skill or team work, it is a sight worth going a long way to behold. I never saw a hitch, and I think I saw fully a dozen goitre operations. I never saw Dr Crile mop the wound, and I have seen a whole operation done without his touching a Spenceer Wells. He just cuts and cuts, and when he has finished the thyroid is separated out, isthmus and both lobes—save for the small posterior shewing of the lateral lobes to which reference has already been made. The vessels are tied off with eight chief's by transfixion. In all the operations for toxic goitres the wound is left open and picked being sewn up in from twenty-four to forty-eight hours. There are three reasons given for this, viz (1) That the septic exudate from the raw thyroid surface is toxic. (2) There is less pain. and (3) That it shortens the operation.

time. Going round one morning with Dr Crile, I saw a patient thirty six hours after operation, and twelve hours after the wound had been sewn up, she was comatose. The stitches were promptly taken out and the wound packed open. I saw her again next morning, and though still looking ill, she was quite conscious and could talk.

I was told that in the first six months of the year (1921) there were performed 2413 major operations of all sorts in Dr Crile's clinic, with a total mortality of 2 per cent. This included all emergencies and bad cases. The mortality of all goitre operations is 1½ per cent.

The little I have to say about thyroxin I learned from Dr Boothby. It is the active principle of the thyroid gland, first separated out by Dr Kendal of the Mayo clinic. The amount found in the normal gland is about 14 milligrammes. It wears away at the rate of about half a milligramme a day, but to replace this loss it is necessary to give one to one and a half milligrammes a day. When injected it takes twelve hours to act at all, and does not reach a maximal physiological effect for five days. Knowing this, the Mayo clinic is opposed to the ordinary method of starting with small doses of thyroid and working up to a large one. It rather advises an opposite procedure.

In conclusion, it should be remembered that this paper is the result of first hand experience and is entirely devoid of any result of book-study. Whatever virtues it possesses, I very willingly ascribe to those to whom such an acknowledgment is due to the workers of the Mayo clinic, and especially to Dr Boothby, who gave time and trouble to the stranger within the gates, and to Dr Crile, at whose clinic I saw so much of what modern surgery can be.

A METHOD OF LIGATURING THE FIRST STAGE OF THE LEFT SUBCLAVIAN ARTERY FROM BEHIND.

By ARNOLD K. HENRY, Dublin

WHILE engaged recently in investigating another problem of the upper thorax upon the cadaver,* I came almost by accident on a relatively simple method of ligaturing the first stage of the left subclavian artery. After demonstrating this method upon several occasions in the School of Anatomy of the Royal College of Surgeons in Ireland, I found that another posterior approach had been used by Sherrill in 1910 and published by him in 1911†. Only 21 cases of ligature of the left subclavian in its first stage are on record, and 7 of these were performed since Sherrill's operation, which is the solitary instance of a posterior approach to this forbidding artery‡.

The anterior approach to the left subclavian is notoriously difficult, a formidable array of nerves and vessels screen the artery. Through these but narrow access is gained even after resection of the inner end of the clavicle, the first rib cartilage, and part of the manubrium. In actual practice, too, the upward bulge of an aneurysm into the neck will not simplify the surgeon's task. The posterior route has the merit of simplicity, and surgery advances through simplification to security. I thus venture to describe in detail the route upon which I chanced.

The transverse process of the second dorsal vertebra, and three inches of the second rib, measured from its head, were removed from the left chest of a hunchbacked cadaver after carefully separating the rib from the parietal pleura. In effecting this separation the pleural dome was slightly depressed, and the first stage of the left subclavian artery appeared in the field. Further separation and depression of the pleura exposed the artery from its point of origin at the aortic arch to the first rib, and definition of all its branches except the thyrocervical trunk was easy. These structures were rendered surprisingly superficial by the kyphotic deformity of the back. Examination of normal subjects showed that in them the left subclavian artery and its branches are further from the dorsal surface of the trunk. The first stage of the artery, however, is just as easily tied in spite of the depth at which it lies, for once the lung and pleura have been depressed, the artery, except for a delicate sheath, lies naked in the thoracic cavity, and is immediately accessible. There is no barrier of vein or nerve, the vessel is directly under the finger. With a suitable needle it is easy to pass a ligature round the artery, and at my request this was done by students who had never previously tied any vessel in the body. Before describing the steps of the operation, certain anatomical points must be dealt with.

ANATOMICAL CONSIDERATIONS

The Muscular Planes—The part of the second left rib which is removed lies between the scapula and the vertebral spines. It is concealed by muscles which anchor the scapula to the vertebrae. The trapezius is spread over the rhomboids, which cover the upper serratus posterior. Division of these muscles allows the surgeon to widen the space

* Posterior Route for Excision of the Cervico-dorsal Ganglion of the Sympathetic. (Section of Surgery Irish Academy of Medicine in Ireland, April 28, 1922.)

† Sherrill raised a flap of skin and muscle and removed about three inches of the 2nd, 3rd and 4th ribs. After pushing aside the pleura, the artery was exposed at the level of the 4th dorsal vertebra as it left the aorta.

between the scapula and the spine, and it is essential that the transverse width of the wound should be as great as possible

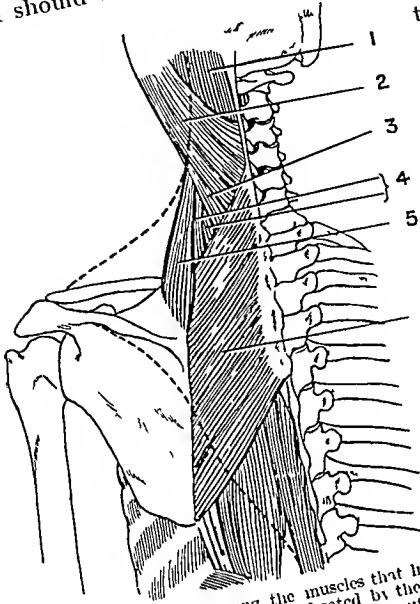


FIG 289—Showing the muscles that lie deep to the trapezius which is indicated by the dotted line (1) Complexus (2) Splenius capitis (3) Splenius cervicis (4) Cervical extensions of erector spinae (5) Levator scapulae (6) Rhomboids

cervical extensions of the erector spinae (the rhocostalis cervicis and the longissimus cervicis) Reckoning from this landmark, the surgeon finds the second transverse process and the second rib *

The Left Subclavian Artery—The anterior relations of this artery in its first stage make an impressive list. Deep to the muscular planes consisting of the sternomastoid, sternohyoid, and sternothyroid, lie the left innominate, internal jugular, and vertebral veins, succeeded by the vagus and phrenic nerves, the carotid artery, and branches of the cervical sympathetic. In the posterior approach however, when the pleural dome has been depressed only one minute structure intervenes between the surgeon and the artery, the ansa subclavaria of Vieussens. This tough but slender loop crosses the back of the vertebral column as the vessel arches after giving off its vertebral branch (Fig 291). The depth of the proximal part of the artery from the dorsal surface is about three inches, that is to say if the index finger could be thrust through the skin it would just touch the artery. By making a large flap as described below, the thickness of the skin and subcutaneous tissue is eliminated from the field, and suitable division of the muscles to the shoulder girdle

Deep to the muscles of the shoulder girdle, the splenius spreads upwards from the dorsal spines, and lateral to the splenius are the cervical extensions of the erector spinae (Fig 289)

The Second Rib—The second rib must be accurately identified. It is not difficult to miss take it for the first, and thus in error to remove the third rib. The second rib and transverse process viewed from behind lie dorsal to the first, and the body of the first rib runs almost directly forwards from the costo transverse articulation, it is difficult to palpate. When, however, the trapezius and the other muscles passing to the scapula have been divided and retracted, the first rib can be felt by hooking the finger deeply down along the neck.

The first dorsal transverse process is a good landmark (Fig 290) it lies at the level of the 7th cervical spine, two finger breadths from the middle line. It is the first transverse process to project beyond the edge of the splenius. Here its tip is felt but is not seen, being covered by two

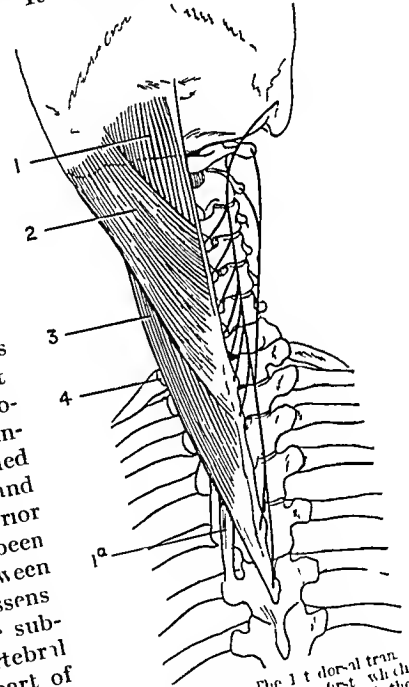


FIG 290—The 1st dorsal transverse process is the first which projects beyond the edge of the splenius. It forms a deep landmark for identifying the second rib (1) Complexus (2) Splenius capitis (3) Splenius cervicis (4) 1st dorsal transverse process (5) 1st rib

* For additional security the second rib should be localized by radiography before operation and the radiologist should be asked to examine the thoracic inlet for accessory cervical or rudimentary thoracic ribs which might confuse the surgeon approaching them from behind

LIGATURING THE LEFT SUBCLAVIAN ARTERY 369

allows the surgeon to work from the plane of the thoracic wall. The 'working depth' of the artery is thus reduced to two inches, which is the actual depth of the artery from the upper border of the manubrium in front. The 'working depth', therefore, is the same whether the approach is from the front or back.

The Left Vagus—The presence of the left vagus need not be feared. The relations of the nerve depicted in most textbooks of anatomy are those which it

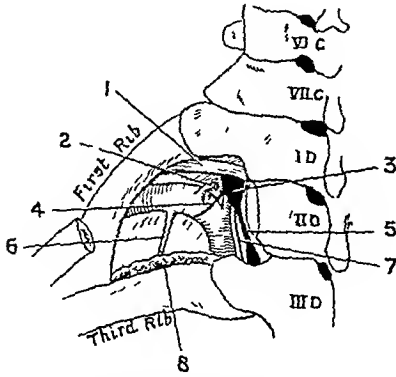


FIG 291.—The relations of the first stage of the left subclavian artery seen from behind after removal of the 2nd dorsal transverse process and part of the 2nd rib. The oesophagus and the thoracic duct which crosses its left side, are not shown in the figure. They are nearer the middle line. To see the origin of the subclavian artery from the aortic arch and to tie the proximal part of the artery the surgeon stands opposite the head of the table. (1) 1st dorsal nerve. (2) Costo-cervical trunk. (3) 1st dorsal ganglion of sympathetic cord. (4) 1st dorsal transverse process. (5) 1st dorsal nerve. (6) Internal mammary artery. (7) Inferior cardiac branch of sympathetic. (8) Pleural dome retracted downwards.

issues after it has been freed by dissection. It then falls away from the common carotid, and lies close in front of the first stage of the subclavian. If this were its true position, it would be in danger when the artery was tied from behind. Actually the nerve is, as Charpy² states, a satellite of the common carotid, and passes downwards and inwards close along this artery, coming gradually forwards as it descends. It thus lies beside, rather than behind, the left carotid at the root of the neck, and is on a plane anterior to the left subclavian (Fig 292). About a finger-breadth above the

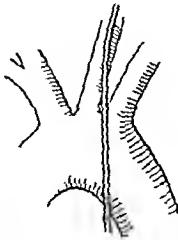


FIG 292.—Diagram showing left vagus related to the left carotid and left subclavian arteries like the oblique stroke of the letter N. The vagus is safe from inclusion when the left subclavian artery is tied from behind.

Further there is a barrier between the vagus and the subclavian which protects the nerve from inclusion when the artery is tied by the posterior route. This barrier consists

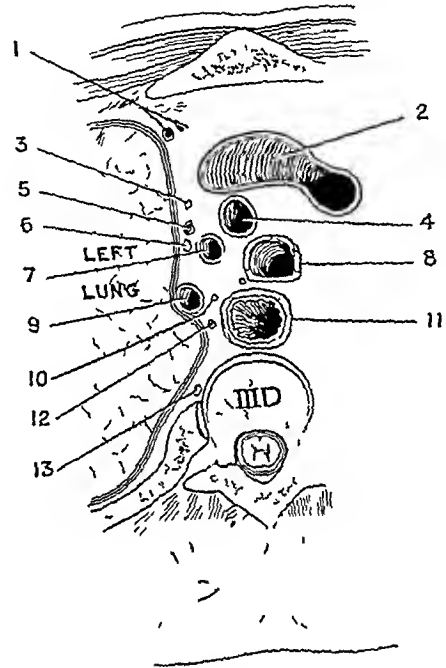


FIG 293.—Cross section through 2nd dorsal vertebra and upper part of manubrium, showing relations of left subclavian artery. Note that the left vagus is still a satellite of the common carotid, the thoracic duct adheres to the oesophagus and is in no danger of injury. (1) Internal mammary vessels. (2) Left innominate vein. (3) Phrenic nerve. (4) Innominate artery. (5) Left superior intercostal vein. (6) Vagus nerve. (7) Left common carotid. (8) Trachea with left recurrent laryngeal nerve. (9) Left subclavian artery. (10) Inferior cardiac branch of cervical sympathetic. (11) Oesophagus. (12) Thoracic duct. (13) Sympathetic cord lying in front of costo-vertebral joint between rib and vertebral body.

aortic arch the direction of the vagus changes abruptly, the nerve passes out, down, and back, to cross the root of the left subclavian artery, so that this part of the vagus lies between the vertical carotid and subclavian origins of the left side like the oblique stroke of the letter N (Fig 293).

of a layer of areolar tissue which contains (1) The middle and sometimes the superior cardiac branch of the cervical sympathetic, (2) Descending œsophageal and tracheal branches of the inferior thyroid artery, (3) An occasional thymic tributary of the vertebral vein. These structures not only make it difficult to expose the subclavian from in front, but obscure the origin of the vertebral artery, which tends to lie at a relatively low level on the left side.

The Left Vertebral Artery—This vessel during development is often absorbed into the aortic arch, and seen from in front may be mistaken for the subclavian, since it then arises from the arch between the left common carotid and the subclavian trunk. This error will not be made in ligation from behind.

The Thoracic Duct and the Inferior Cardiac Nerve—The thoracic duct will not be injured from the back, it stripes the left side of the œsophagus in the superior mediastinum, and only leaves it to pass in front of the root of the vertebral artery. In an anterior attack the duct, though not in contact with the first stage of the subclavian, may be injured as it arches outwards over the vertebral origin.

The inferior cardiac branch of the sympathetic also lies medial to the artery. With the most ordinary care it is easily avoided (*see Fig 291*).

TECHNIQUE OF OPERATION

A good head-light should be used, but in the cadaver I have repeatedly tied the artery without artificial illumination. The patient should lie prone, with the left shoulder clear of the table and the left upper limb hanging vertical (*Fig 294*). Make the upper dorsal region as kyphotic as possible. This gives the space between the scapula and the vertebral column its maximal width.

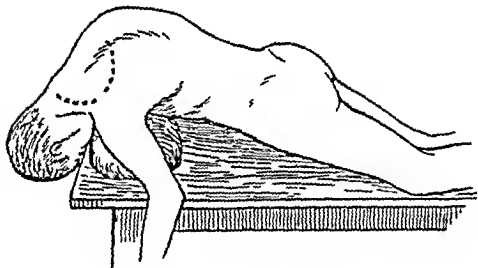


FIG. 294.—Showing skin incision and position securing maximum abduction of the scapula.

1 Find the 7th cervical spine. Mark (a) A point four finger-breadths above it and one finger breadth to the *right* of the middle line. (b) A similar point six finger breadths below the 7th spine, (c) A point over the middle of the spine of the left scapula. Join these three points by the incision shown in *Fig 291*, which is carried down to the sheath of the trapezius muscle. Raise the flap of skin and subcutaneous

tissue thus outlined and turn it over to the *right* of the middle line.

2 With a vertical cut one finger breadth to the left of the vertebral spines, divide the origins of (a) the trapezius, (b) the rhomboids, and (c) the serratus posterior superior. Do this first at the middle of the wound where the silvery tendon of the serratus indicates the depth reached. Extend this incision throughout the entire length of the wound. Retract the divided muscles outwards. The pointed caudal end of the fleshy splenius is now exposed.

3 At the level of the 7th cervical spine and two finger-breadths from the middle line, find the tip of the first left dorsal transverse process, remembering that it is the first which projects beyond the edge of the splenius. Find the second left rib.

4 Clear the transverse process of the second dorsal vertebra as far as the lamina. Clear at least three inches of the second rib. Divide the transverse process at its root and remove it. Divide the rib as far as the wound will permit from the costo-transverse articulation.

5 Raise the proximal cut end of the rib. With finger push the pleura away from its head and neck. Rotate the rib segment and divide its attachments. The sympathetic cord is now seen close to the vertebral body lying on the pleura like a tape.

6 Very gently push the pleural dome downwards and outwards from the vertebra.

A small strand will now be found holding the pleura to the neck of the first rib. This strand is a branch of the superior intercostal artery. Divide and tie it. The pleural dome can then be freely depressed, and the left subclavian is felt by the finger passed vertically and at a tangent to the vertebral body. *The removal of the transverse process, together with the costal neck, permits of this direct approach.* A broad malleable retractor keeps the lung and pleura out of the field. It should be polished so as to reflect light into the cavity. The artery is isolated under direct vision by blunt dissection and its sheath is opened in the usual manner, using a long dissecting forceps. The ansa subclavia should be avoided.

7 The surgeon stands facing the head of the table. An aneurysm needle with a slot eye is passed with the left hand from within outwards. Introduction of the right forefinger into the wound facilitates this manoeuvre. The eye is threaded with a ligature, or with a guiding thread to which a definitive ligature (a tape, for example) is attached. Ample space is afforded for securing the knot.

The internal mammary and costo-cervical trunks can be tied at their origins. The vertebral artery is obscured by the cervico-dorsal ganglion of the sympathetic, but can be safely ligatured by opening the subclavian sheath close to the vertebral origin and passing an aneurysm needle round the parent trunk so that its point appears in the angle between the subclavian and the vertebral artery. The thoracic duct may thus be avoided. The thyro-cervical trunk is difficult to secure by the posterior route.

My best thanks are due to Professor E. J. Evatt, D.S.O. for the many opportunities he has given me of testing this method on the cadaver. For the figures illustrating this paper, I am indebted to the text-books of Cunningham, and of Poirier and Charpy from which they have been modified.

SUMMARY

An approach to the first stage of the subclavian artery is obtained by costo-transversectomy at the level of the second rib on the left side. Depression of the pleural dome leaves the artery naked from the aorta to the first rib, no structure (excepting the ansa subclavia) intervening between the operator and the vessel.

The first stage of the vessel can be ligatured in any part of its course, and its branches, except the thyro-cervical trunk, can be tied with relative ease.

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 - ² *Traité d'Anatomie*, 2nd ed., vol. II, pt. 1, p. 426.

THE PLACE OF OPERATIONS FOR SPINAL FIXATION IN THE TREATMENT OF POTT'S DISEASE

By G. R. GIRDLESTONE, Oxford

IN 1919 I published a paper¹ on 50 cases in which operative spinal fixation had been performed by my colleagues or myself. I have now particulars of 50 additional cases and place in the treatment of Pott's disease critically assessed. In the past, operation has too often been thought of as if it did in itself definitely provide a short cut to the cure of Pott's disease. Wholly extravagant claims have been made on behalf of this new means of treatment, and altogether too much reliance has been placed upon it, cases have been treated by operation without proper splintage, without open air methods, and without adequate rest or sufficient time. This mistaken view of the scope of operative fixation has led to many failures and much disappointment. As a result the operation has not been seen in true perspective. It is highly esteemed by those who use it as "part of the conservative treatment of Pott's disease."²

There can be no short cuts to cure in this disease. End-results teach us that in serious bone and joint tuberculosis short cuts lead altogether in the wrong direction. In the spinal lesion the gradual processes which end in ossification take their inevitable time. And this lesion is only part of an invasion of the patient's body by the tubercle bacillus, he is mortally attacked, for the time being the invader is gaining ground and the victim exhausted. Every influence to restore and raise his vitality must be applied. His needs are twofold, and before treatment is complete: (1) His general health and powers of reaction must have risen superior to the destructive activities of the tubercle bacilli, in his spine, and elsewhere; (2) The spinal lesion must be healed, and sound structure in the splintage of the damaged part re-established. Operative spinal fixation may help it cannot replace the established principles of treatment.

Rationale of Operations for Spinal Fixation—The object of the various methods of operative fixation of the spine is to promote bony union between the dorsal elements of the vertebral column and so to provide permanent internal splintage of the affected part.

Apparently Albee's plan of graft fixation³ grew out of comments that Brackett had made on the natural ossification between and round the spinous processes which occasionally occurs in the course of Pott's disease.⁴ This interlaminar and interspinous ankylosis occurs very slowly, and only after extensive destruction of the bodies has led to crowding together of the laminae and spines. It was clearly desirable, if practicable, to promote bony union between the spines or laminae before rather than after, destructive lesions had taken place.

Libb's⁵ states the reasons that induced him to develop and carry out his method of operative fixation thus: "It was felt that absolute elimination (of motion) would hasten the cure and perhaps prevent deformity. The absolute elimination of movement in any joint is accomplished only when the bones entering into its formation become fused. This is sometimes accomplished by nature without operative aid. In the case of a vertebral joint affected by tuberculosis however, nature is too slow." In the **The Status of the Spine in Relation to Caries**—A review of the mechanical problem involved by destructive lesions of the vertebral bodies is an essential preliminary to an intelligent discussion of the value of the operations designed to promote spinal fixation.

Spinal mechanism is very complex, but in relation to caries it can be considered in a simplified form. The spine may be represented as composed of three units: (1) Anterior (somatic) column—centra and discs, (2) Lateral columns—articular and other lateral processes, (3) Dorsal processes—laminae and spines. The bodies contain much of the spongy bone and red marrow beloved of the tubercle bacillus, whereas the lateral columns and dorsal processes are formed of comparatively hard bone, which is seldom attacked and resistant to destruction. The weight of the body is normally carried both by somatic and lateral columns*. The dorsal processes form a system of levers which are moved or held by the erector spinae muscles; they carry no direct weight whatever, but act purely as levers working on the fulcrum furnished by the lateral articulations. The movements of flexion and extension are controlled by the lateral columns, though the axis of movement does not necessarily pass through the lateral articulations.

When caries causes extensive loss of substance in the somatic column two processes are likely to take place: (1) Telescoping, (2) Inflexion.

1 *Telescoping* is a sliding of the lateral articulations, and 'crowding together' of the dorsal processes, to correspond with the degree of somatic loss. Now this movement is limited by the shape of the lateral articulations and by imbrication of the dorsal processes. It is especially free in the lumbar region. When telescoping has reached its limits and somatic destruction still continues, another and entirely distinct process comes into play unless the posture of the spine is artificially controlled by effective splintage. This second process is—

2 *Inflexion*, i.e., a 'falling forward' of the segment of spine above the gap until the body above is once more supported. If there is not much destruction, the under surface of the centrum above the gap meets the upper surface of that below. But if there is extensive loss of bone substance, it is the anterior surface of the body above the gap that will be opposed to the upper surface of the centrum below. This extreme and strictly localized flexion (Menard's 'complete inflexion') involves a subluxation of the lateral articulations, with the result that the lowest lateral articular surface of the upper segment rests on the 'knife edge' of the upper lateral articular surface of the segment below the gap. This is a most unstable condition, and represents the so-called 'pathological fracture dislocation' of spinal caries.

If, however, when telescoping is complete the spine is firmly maintained in the extended position, the pressure is taken entirely on the lateral columns, an actual gap in the bony continuity of the somatic column being left. Now this local extension of the spine can sometimes be maintained in recumbency by careful posturization, but it cannot be maintained by any external splintage when the patient gets up†. Usually there is gradual development of 'complete inflexion' (Menard). All that ambulatory splintage can do is to ensure the development of sufficient compensatory curves just above and below the lesion, so that the hump may be minimized and erectness of the spine as a whole may be restored.

It was hoped that the production of a synostosis of the dorsal processes, and the maintenance of approximation of these posterior levers, would achieve a permanent local extension.

What can Successful Spinal Fixation Actually Achieve?—

1 The operations of so-called 'spinal fixation' are in no sense radical; they do not aim at or encompass any extirpation of the diseased parts.

2 They are directed toward fixation of the dorsal units only, and should really be termed 'posterior spinal fixation'. The dorsal units do not carry weight, but consist of a series of levers connected by, and acting through the lateral columns.

3 Successful posterior spinal fixation produces a series of bony bridges uniting and holding a succession of these levers. This artificial synostosis of the dorsal units is only

* Throughout this description for the sake of simplicity the spine is supposed to be erect.

† Cases of definite local bone destruction producing a gap in the somatic column are being discussed.

sufficient to prevent flexion of the segment if the lateral columns present a series of fulera. The lateral columns then actually carry the weight, while the graft (or osteoplastic connection) maintains the approximation of the dorsal levers and stabilizes the upper and lower segments at their junction on the fulera.

4 The union between the dorsal units is not designed to stand a cross breaking strain. Provided the lateral columns furnish the fulera, the tendency of the upper segment to fall forward at the site of the lesion will throw a strain consisting mainly of vertical tension on the graft (experimentally shown by Calve and Gulland, de Quervain and Hoessly, and others).

5 Posterior spinal fixation, since it acts by leverage and stabilization and does not produce direct sustentation, is a form of splintage. It helps to protect, but cannot replace, the natural process of healing. Spinal fixation may play a useful part in the organized provision of rest and time. Successfully achieved, and in suitable cases, it aids the rest and shortens the time, but cannot replace either.

6 Spinal fixation offers a further effect in preventing deformity if it suffices to maintain local extension of the diseased segment after the patient gets up. This depends on (a) The strength and stability of the lateral columns, (b) The protection of the new bony tissues from strain until they are strong enough to stand it without giving way. For the latter, adequate splintage, the stimulus of gradually increased function, and time are necessary.

Various Methods of Posterior Spinal Fixation—A number of methods have been devised in order to achieve this end. The spinous processes have been wired together, but the wires gradually cut through the bone. Calot had suggested the turning up and down of periosteal flaps from the spinous processes, and Hibbs developed and extended this method, turning up and down flakes from the laminae, and producing interspinous union by the partial section and fracture of the spinous processes.

Albee used a graft from the tibia. De Quervain, independently, and only slightly later, used a graft from the spine of the scapula (having previously applied this method in 1911 in the treatment of fracture dislocation of the spine). These writers quote Ombredanne as having used the median border of the scapula, Toblase as using a portion of rib. More recently Gallie and Robertson⁶ in Canada showed experimentally on young dogs that a boiled graft could be used with as good success as a fresh one. They mention having applied the method of grafting successfully in 60 cases of spinal disease. Further possibilities were opened by the experiments of Hey Groves and others in the use of boiled beef-bone grafts.

While the whole subject of the share that the actual transplanted bone cells take in the rebuilding of the graft cannot be discussed here, the animal experiments of both Gallie and Robertson, and de Quervain and Hoessly, show how rapidly an autoplastic graft (boiled by the former, fresh in the latter's experiments) is invaded by the bone cells of the part. The time depends on the density of the bone inserted, in the ulna or rib it is about three weeks.

De Quervain and Hoessly partially excised the body of a vertebra and inserted into the split spines an autoplastic graft from the ulna. They found (1) That this prevented the formation of a kyphos, (2) That the graft was firmly healed in place in two months, (3) That the segment of vertebral column fixed by the graft, when removed from the body, supported a pressure of 40 kilo applied to the centrum above the defect. Control dogs on which the same partial removal of bodies was carried out without grafting developed a kyphos.

Calve, in his most interesting paper, which has already been quoted,⁷ on the anatomical and pathological changes in the spinal column from the point of view of destruction, displacement, healing, and structural stability, discusses the methods of Albee and of Hibbs as instances of the graft versus the osteoplastic method. He quotes Albee's operation as being eminently satisfactory and practical in the lumbar region, but very difficult to apply in the dorsal region on account of the kyphotic deformity. A strong straight graft will not fit into place, and multiple partial cross sections make the

graft weak and liable to fracture at the point of the angle where the strength is most needed. He criticizes Hibbs' method in that he has found it very difficult to perform, and when he has performed it, has felt dissatisfied with the prospects of achieving a strong bony union. Calve himself advocates a method which includes the removal of the spines, the exposure of the laminae, and the preparation of a broad, raw, osseous surface to which he applies the graft. This is an extensive operation, and one involving the loss of valuable structures.

De Quervain and Hoessly, on the other hand, point out the virtue of a graft method over an osteoplastic method in that the fixation of the spine is achieved so much more quickly and certainly in the former. The difficulty of fitting the ordinary tibial graft to a kyphotic angle has been surmounted by the use of a previously prepared, boiled, human or bovine graft.

The writer agrees with the expression of opinion of Calve that the Hibbs operation is exceedingly difficult to complete with any certainty of producing a solid union throughout the region affected and the several segments above and below, and further, that it takes longer and involves more hæmorrhage than the graft method of Albee. But those who have seen Hibbs himself perform the operation bear witness to the thoroughness of his achievement and his wonderful realization of a bloodless field.

While the Commission which has been investigating the end-results of the various methods of operative fixation in America is apparently in favour of the operations carried out by Hibbs and his assistants, two factors must be considered before one can apply their findings to the work in this country. (1) The Hibbs operation is very difficult, while the Albee operation is easy except where there is much kyphosis. (2) Albee, in his earlier cases at least, seems to have considered that the implantation of the graft eliminated the need for splintage, and allowed his patients to get up without supports within 6 or 8 weeks of the operation, clearly this must have prejudiced his results very seriously. While the writer has had considerable experience of the graft operation, he has little comparable knowledge of the osteoplastic method. Fortunately Hibbs⁵ himself has published a detailed account of his operation and a report as to the end-results of 210 cases which had been submitted to operation between January, 1911, and January, 1915, i.e., three years or more before his report. Although the account of the operation given in this paper is very graphic and should be read by any surgeon who is proposing to carry out the method, the writer feels that the technical details of the subperiosteal dissection and exact adjustment of the tiny portions of bone raised from the laminae are so intricate, and withal so essentially important, that in order to carry out Hibbs' method in its entirety it is probably necessary to see him or someone who has worked with him, operate. The whole point of this operation depends on the formation of a strong complete posterior plaque of bone, and any incompleteness of interspinous and interlaminar union would lead to failure. In Hibbs' hands the results are extraordinarily good, as the tables on the following page show.

Photographs, radiographs, and tracings were made periodically in each case. Hibbs found that deformity had increased in 18, decreased in 17, and had remained unchanged in 139 cases.

Of 35 cases suffering from cord pressure (26 paralyzed, 9 slightly spastic with increased reflexes), 30 were cured, 2 remained paralyzed, 3 died.

He had no operation mortality, and he states that there was hardly any shock, and that all the wounds healed by first intention. There was no selection of cases, except that the presence of a discharging sinus in or near the field of operation was taken as a contraindication. Every patient who would consent and whose general condition warranted the anaesthetic was subjected to operation. In only four cases had fusion failed.

One interesting sidelight emerges from this work. Hibbs' operation involves a thorough clearing of the spines and the laminae right out to the base of the transverse processes, exposing the lateral articulations. He was thus in a position to observe involvement of any of these parts, and in only 5 cases was there disease of spines or laminae.

This is most interesting in its bearing on the natural processes of interlamina and inter-spinal ankylosis. Can the crowding together of laminae and spines alone produce inter-union? It is difficult to imagine that this union would occur between normal periosteum covered portions of bone. The question is not answered by the segments of spinal column showing dorsal ankylosis in Pott's disease which are to be found in pathological museums, for it is often the most remarkable and unusual specimens that are preserved.

Table I—HIBBS' OPERATION RESULTS
IN 210 CASES

RESULT	NO OF CASES
Cured	157 (75 %)
Doubtful	22 ¹
Dead	31-

¹ 2 have remained paralyzed though the fusion is complete and there is no evidence of active disease.

² 13 died of miliary tuberculosis, 3 of tuberculous meningitis, 1 of phthisis, 3 of amyloid disease.

Table II—CURED CASES AGE AT TIME
OF OPERATION

AGE IN YEARS	NO OF CASES
1-2	2
2-4	27
4-6	22
6-10	47
10-15	43
15-20	6
20-30	5
30-40	3
40-50	2

Table III—CURED CASES NUMBER OF
VERTEBRÆ INVOLVED

NO OF VERTEBRÆ	NO OF CASES
2	19
3	35
4	23
5	32
6	20
7	14
8	7
9	3
10	2
11	1

Table IV—CURED CASES REGION IN-
VOLVED

REGION	NO OF CASES
Cervicodorsal	6
Dorsal	71
Dorsolumbar	50
Lumbar	28
Lumbosacral	2

REPORT ON 100 CASES

I have collected particulars of 100 consecutive cases of spinal caries in which operations for spinal fixation have been done by my colleagues or myself at the Shropshire Orthopaedic Hospital* or the Headington Orthopaedic Hospital in the period 1914-1921. In 95 cases a graft, usually from the patient's tibia, has been applied to the split spines according to Albee's method, or to the bared laminae. Personally I favour the former. In 5 cases osteoplastic methods were employed. In all but the very earliest cases either a mechanical or motor saw has been used.

Operative Mortality—Two cases died within a week of the operation. Of these, one, a child of 5, died from shock within twenty-four hours; post mortem examination showed that the graft had been placed too deeply, was projecting through the laminae and pressing on the cord. The other case, a boy of 14, died four days after operation with abdominal pain and hyperpyrexia; the spinal wound was clean, no post mortem examination was allowed.

In first 50 cases	2 died
In second 50 cases	none died
Operative mortality = 2 per cent	

Later Mortality—Six cases are known to have died since—

Case aged 34	died 4 years later from general tuberculosis
" 28	4 years later from pneumonia
" 6	5 months later from general tuberculosis
" 17	1 year later from shock under anæsthetic during manipulation of hip
" 34	1½ years later from continued caries
" 9	1 year later from tuberculous meningitis

* I acknowledge gratefully the courtesy of Sir Robert Jones M. Aitken and Mr Dunn and the help of Mr Noble who will, I hope, publish a full account of the Shropshire cases later.

Age Incidence —

AGES	FIRST 50 CASES	SECOND 50 CASES	TOTAL
Under 16	36	16	52
16 or over	14	34	48

Abscess before Operation —

Abscess palpable but not discharging	24	Clean cases	94
Abscess not present or not recorded	70		
Discharging sinus or sinuses	6	Septic cases	6

End-Results — Recent reports (December, 1921, or later) of 59 cases grafted 1914–1918 —

Well	42
First heard of well, but no recent report available	5
With signs of recurrent or persistent disease	4
Dead (see particulars given above)	8

Recent reports (December, 1921, or later) of 41 cases of 'fixation' done 1919 or later —

Healed and doing well	36
Discharging psoas abscess, but doing well	1
With signs of recurrent or persistent disease (3 of these were previously septic, the other paraplegic)	4
Dead	0

The later results are better than the early ones. This may be due to the following factors: (1) We now always operate in the turning case (see figures), (2) We keep the patients longer on their frames in the open ward before letting them get up and leave hospital, (3) Our after-care of cases is now more complete.

After treatment — From the first it was realized that graft fixation could not be expected to replace splintage, and that the firm fixation of the graft should not be relied upon for at least three months. The rule adopted was that the case should be treated on a frame for three months subsequent to operation, and then on a spinal support in bed for a further month. It has been found advisable to extend frame fixation in children to six months or more, and in adults to four months, and a spinal support is worn for a year or more after the patient is allowed to get up.

The Turning Case — This apparatus plays an important part before, during, and after operation in every case. It is essentially a removable interior plaster-bed (Fig 295) it extends from chin to ankles. One of these is prepared for every case of spinal cases that is treated on any form of frame or plaster bed. It is made with the patient lying on the frame or in the plaster-bed and is arranged so that it fits accurately over the whole body of the patient, including the legs as far as the ankles, and, in the case of cervical and high dorsal disease it extends to the chin and mastoid processes. It is always kept near the patient's bed and is used whenever he has to be turned over for any purpose (Fig 296) for washing for heliotherapy or for dressing a wound. If an operation is to be carried out, the turning case is used for the preparation, during the performance, and for the after-dressings of the operation, and the patient lies in the turning case with the frame removed during the operation (Fig 297).



FIG 295 — Turning case

When the turning ease is to be used for operation additional bars are attached to the front of the plaster running up on each side of the head (Fig 298). A strap is put across between these, to support the forehead, thus leaving the face held free for the anaesthetist's mask.

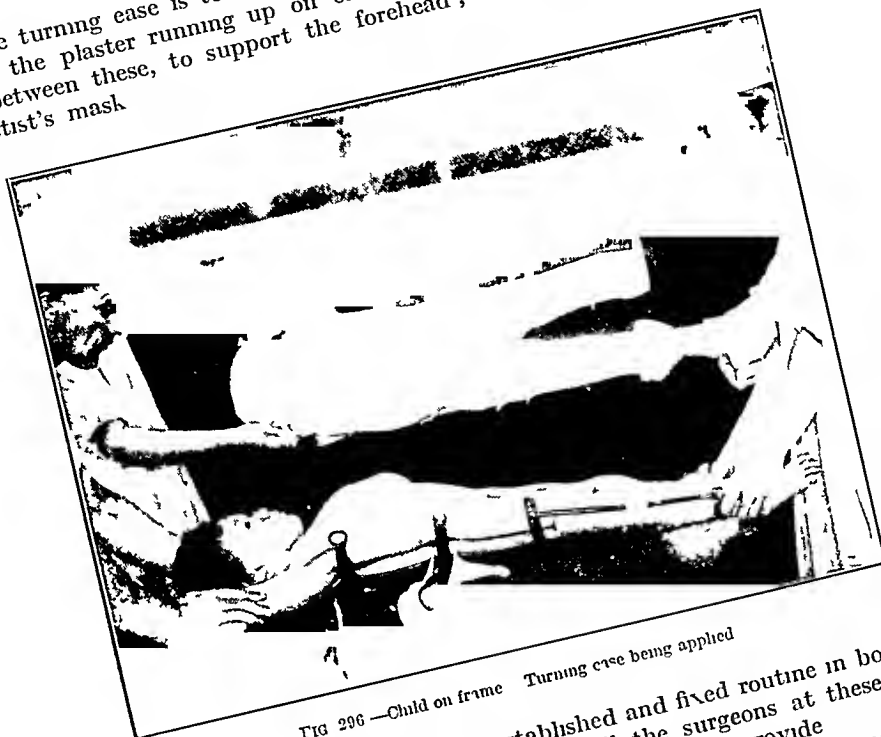


FIG 296—Child on frame Turning ease being applied

The use of these turning eases is the established and fixed routine in both the special hospitals to which the writer is attached, and all the surgeons at these hospitals are entirely convinced of the great advantage and security they provide.

Their use during operations for spinal fixation has practically eliminated all risk of harmful movement of the spine during the operation, and the patients have certainly suffered less often and less severely from shock.

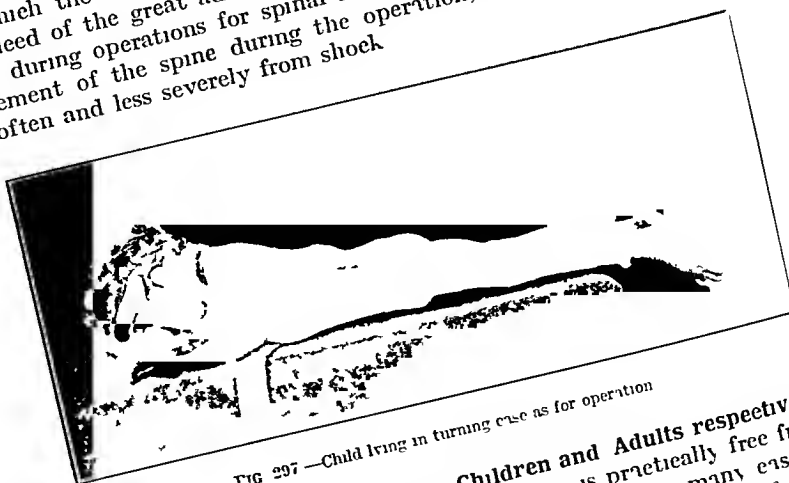


FIG 297—Child lying in turning ease as for operation

Suitability of Operative Treatment in Children and Adults respectively—The end results have clearly shown that in adults the operation is practically free from danger and leads to good structural stability but that in children, while in many cases the graft has persisted in others it has gradually become absorbed. Henderson,⁸ discussing end results of operative fixation (Hibbs and Albee methods), bears out this conclusion. He says: "By repeated x-ray examinations of the spine following transplantation of bone in

children we have seen the graft gradually absorb so that there is no trace of it at the end of a year. The same observation also applies to osteoplastic operations." The younger the child, the more often is the graft absorbed. Further, the death of two children has been associated with the operation, in one case this was owing to too deep an implantation of the graft, and in the other unfortunately death occurred a week after operation from an unknown cause.

In adults, then, grafting is eminently satisfactory, in children much less so. Fortunately, though this limits the scope of the operation, its application is most successful where it is most needed. The main virtues of the operation concern (1) The early production of sound structural stability, (2) The shortening of the period of recumbency necessary. Now in children

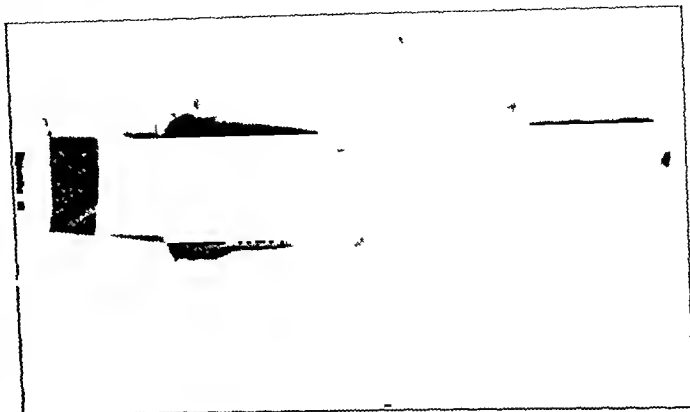


FIG 298.—Turning case fitted with auesthetic extension

structural stability will almost always be realized provided a sufficient length of recumbent treatment in an open-air hospital is given. They thrive, and, while their spinal lesion is in process of consolidation, gradually live down their deep-seated lymphatic tuberculosis. Under the provisions of the Board of Education, the children of school age are at school while in bed in hospital, so that there is no educational reason for hurrying or shortening the period of recumbency. In young children, then, grafting is unreliable and not particularly needed. In adults, on the other hand, structural stability is seldom really safely realized without operative fixation. Adults do not thrive in general health when laid up in bed for long periods, and can seldom spare so long a time without grave dislocation of their work and depreciation of their earning capacity. In adults, then, successful posterior spinal fixation shortens the necessary period of recumbency and helps to produce permanent structural stability, and in adults both the shortened period of hospital life and the realization of structural stability are of out-standing value.

CONCLUSIONS

Operations for spinal fixation are in no sense radical. They concern the splintage and permanent stability of the affected part of the vertebral column. The sound healing of the spinal lesion and the effective resistance of the body to tuberculosis must be awaited before treatment is complete. External splintage of the spine must be maintained continuously before operation during the operation, and afterwards until firm stability of the affected section is assured by the restoration to soundness of the bones and ligaments of the part, coupled with the strong hold of the graft or osteoplastic union.

In adults posterior spinal fixation is reliable, and has great value as "part of the conservative treatment of Pott's disease." In young children it is less reliable, and at the same time less needed and is therefore seldom, if ever, indicated.

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PHOSPHORUS NECROSIS OF THE MANDIBLE

BY H P PICKERILL, NEW ZEALAND

'Phossy jaw', as phosphorus necrosis of the jaw is called by factory workers, is becoming increasingly rare, since the use of the poisonous yellow phosphorus has been prohibited by law. The present is the only case I have any cognizance of, in which the 'phossy jaw' was acquired through mixing rabbit poison (phosphorus and pollard, etc.)

The patient, a man, age 60, had been engaged for many years in mixing such rabbit poison in Central Otago, New Zealand. About twelve months ago he began to have pain in a lower wisdom tooth, this was extracted, and he went on with his work. About a month afterwards he was sent down to me with a well marked periostitis and necrosis on the left side of the mandible, which, despite very free incisions and efficient drainage both inside the mouth and under the jaw externally, rapidly spread all round the jaw. The pain during this period was severe, and only morphia controlled it. The acute stage, however, soon subsided, and the tissues settled down to sequestration accompanied by

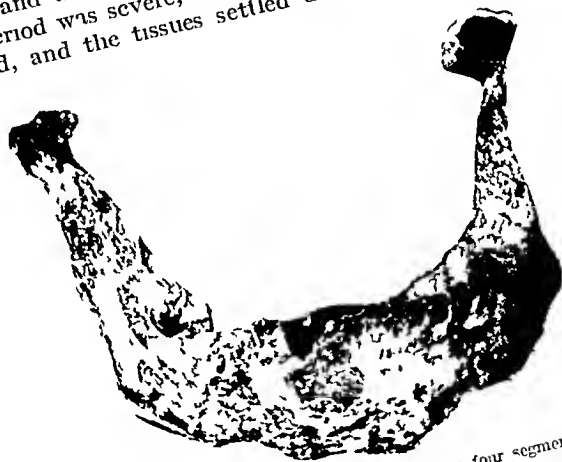


FIG 299.—Necrotic phossy jaw removed subperiosteally in four segments and in two stages

much suppuration, for which free external drainage was provided on both sides of the jaw. The patient's health was maintained at as high a level as possible during this period by a very nourishing diet, tonics, and fresh air. His resistance kept up well and he showed no signs at all of a general toxæmia, which is the cause of fatal termination in such cases.

At the end of six months, judging sequestration to be complete with the development of a considerable involucrum all round the jaw, I proceeded to remove the necrosed bone. This was done in two stages. The left half was removed first in two sections through a long incision below the mandible, the involucrum being split along its lower border to allow of removal of the contained sequestrum, this was accomplished without any very great difficulty. Three weeks later the right side of the jaw was treated in a similar manner, with an equally satisfactory result. It is essential that such cases be left until complete sequestration has taken place, in order to stimulate the formation of new bone, the old method of continual curetting led to disastrous results.

The four pieces of the necrosed mandible, on being put together (*Fig 299*), showed that the sequestrum consisted of a complete mandible—constituting a somewhat unique specimen

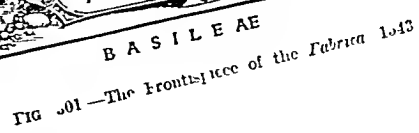
The patient has made an excellent recovery (*Fig 300*) Note the small amount of deformity, and the very fair prominence of the chin This is due to the formation of new bone which is taking place freely For some time—about a fortnight—he had difficulty in controlling his tongue and preventing it falling back into his pharynx, especially at night but when the involucrum—new mandible—united again this trouble ceased

The interest in this case centres around several points —

- 1 The extensive destruction of bone
- 2 The excellent stimulus to new bone formation derived from the presence of the sequestrum
- 3 The advantage obtained by not being in too great a hurry to remove the sequestrum, and especially the avoidance of continual 'curetings



FIG 300—Photograph of the patient after removal of the whole of his lower jaw



VESALIUS: HIS DELINEATION OF THE FRAMEWORK OF THE HUMAN BODY IN THE 'FABRICA' AND 'EPITOME'.

By W G SPENCER LONDON

(The Fourth Vicary Lecture, delivered at the Royal College of Surgeons of England on December 14, 1922)

THE Company of Barbers and the Guild of Surgeons of London, after their union in 1540 made special provision for the instruction in anatomy of the members of the United

ANDREAE VESALII



Fig. 101—Portrait of Vesalius. Fabrica 1543

Company. In commemoration of this period the Barbers' Company in 1919 instituted the Thomas Vicary Lecture, an historical lecture on anatomy or surgery to be given at the Royal College of Surgeons. Sir John Tweedy, past President of the College and past

Master of the Barbers' Company, was the first lecturer. The second lecturer, Sir D Arey Power, Vice-President, has made notable contributions to the history of the Guild of Surgeons. The third Vicary Lecture was given by Sir Charles Ballance on the history of trephining and of brain surgery.

The natural prejudice against the examination of human bodies after death had prevented the art of medicine from developing beyond the stage reached by the Greeks, with the Revival of Learning, however, the human mind became more open to reason, so that a knowledge of structure and of function, also of the changes caused by disease and accident, was obtained by examining human bodies after death, and in addition, anatomical specimens began to be preserved in museums. Supplementary to this was the knowledge acquired by experiments on animals. As the study of human anatomy and physiology increased, it was made the more widely known by the art of printing, there further developed an increased skill in making anatomical drawings which were then reproduced as illustrations by engraving on wood blocks and copper plates.

Thomas Vicary the first Master of the United Barber Surgeons' Company was surgeon to St Bartholomew's Hospital, also to King Henry VIII and his successors, Edward VI, Mary, and Elizabeth, moreover, in 1554 he was appointed surgeon to Philip II of Spain.

At the time corresponding with the painting by Hans Holbein of the famous picture which adorns Barbers' Hall, Vesalius was preparing his great work the *Fabrica*, together with its *Epitome*, both of which were published in the summer of 1543. After that Vesalius succeeded his father as surgeon to Charles V and when that emperor abdicated he transferred his services to Philip. Thus, in quite different ways, both Vicary and Vesalius came to hold the appointment of surgeon to Philip II of Spain.

The year 1914 was the 400th anniversary of the birth of Vesalius and the 350th of his death. It had been proposed to celebrate his memory at the end of that year, but when that time arrived, Brabant, and Brussels, his birthplace were in the hands of the enemy whilst the Library of the University of Louvain, at which Vesalius was educated, had been burnt, together with examples of his works. In neutral Holland, however, especially at the University of Leiden, where Vesalius has always been held in honour several communications concerning him and his writings were published (*Janus*, 1914, Vol. LV). About the same time, in an oration delivered before the German Anatomical Society, Dr Holl of Graz commented on the description of the brain by Vesalius (*Archiv f. Anatomie* 1915, s. 115). In America Dr Cushing and others exhibited and discussed several of his writings. In this country, Mr H M Spielman had in course of preparation an *Iconography* concerning the portraits of Vesalius which the Belgian Government intended to publish, but which were afterwards sent to America (communication by letter).

If a reason for selecting the subject of this lecture need be given, it may be noted that this year there has been celebrated the foundation of the University of Padua where from December, 1537, to August, 1542, Vesalius taught as the first Professor of Anatomy, whilst composing his books.

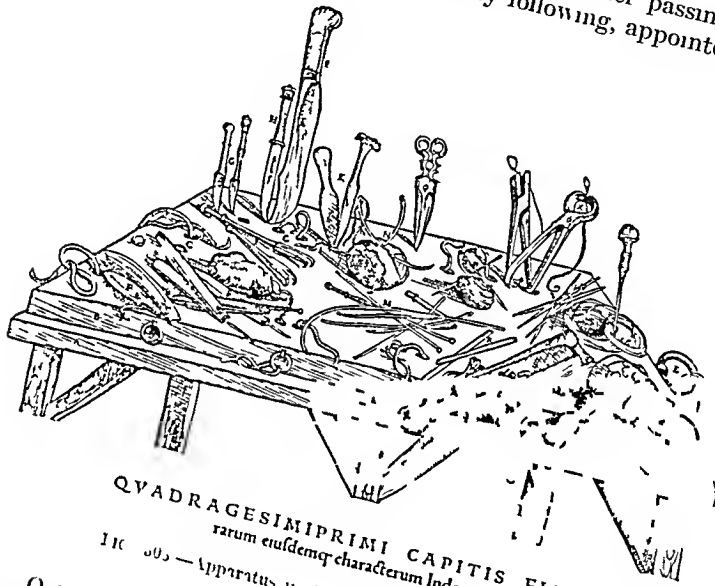
There has accumulated a mass of literature about Vesalius—'Vesaliana'—but all that is well established about him and his illustrations is based essentially upon his own statements—nothing beyond that can be deemed authentic. In 1892 Dr Roth of Basel published an important study of the writings of Vesalius (Dr Roth, *Andreas Vesalius Brucellensis*, Berlin 1892). His enthusiasm, however, led him into some exaggerations which provoked criticism. Since Roth's book, Leonardo da Vinci's drawings have been reproduced and were described by Professor William Wright in his *Arts and Gale Lectures* 1918.

The anatomical illustrations contained in medieval manuscripts can be examined in Professor Sudhoff's Collection (K. Sudhoff, *Ein Beitrag zur Geschichte der Anatomie im Mittelalter*, Leipzig 1908). They consist of diagrams largely imaginary, no better for the most part than the drawings of untaught schoolboys. Of those in printed books the best perhaps, are to be found in Berengario da Carpi's *Commentary on Mundinus*.

VESALIUS

The great Italian artists in the fifteenth century, under the patronage of princes and popes, studied human anatomy by dissection, and made drawings from anatomical preparations. Palluol (1429-98), an elder contemporary of Leonardo, whose Martyrdom of St Sebastian in the National Gallery shows semi-nude male figures, dissected many bodies in the course of examining muscular outlines, and eight drawings by him are in the Louvre. Leonardo da Vinci (1452-1519) stated to Antonio Beati in 1517 that he had dissected more than thirty men and women of all ages, the first date relating to his anatomical studies, and they did not receive attention until he became so well known, he did not publish his anatomical drawings, there is no evidence as to any influence upon Vesalius, and he did not receive attention until recently. Further, Albrecht Dürer (1471-1525), Michael Angelo Buonarroti (1474-1563), Titian (1477-1576), Della Torre (1482-1506), Raphael (1483-1520), Hans Holbein (1497-1543), all to some extent studied artistic anatomy by the aid of human dissection previous to Vesalius.

In December, 1537 Vesalius went to Padua, and after passing the examination for the degree of Doctor of Medicine, was, on the day following, appointed by the Government



QVADRAGESIMIPRIMI CAPITIS FIGV
rarum eiusdemq; characterum Index
FIG. 110 — Apparatus used in dissection by Vesalius

of Venice. Doctor Ostensor et Insector, a post which marked the commencement of a separate professorship in anatomy. Early in 1538 he published a revised edition of the *Institutiones Anatomicae* which Gunther von Andernach had extracted from the writings of Galen. In order to explain to students Galen's statements he issued at the same time his *Tabula Anatomica*. These were distributed as loose sheets and hence have all disappeared except two collections of six sheets each. One collection was reproduced in full size by Sir William Stirling Maxwell in 1874. The other extant collection is preserved in the library of St. Mark's and was reproduced in three quarter size by Dr. Holl of Graz and Professor Sudahoff of Munich in 1920. Thus this early production by Vesalius has been made well known but it should be recognized that the intention was to illustrate Galen's statements. Subsequent to that publication Vesalius extended his knowledge of human anatomy and undertook with greater boldness the correction of Galen's statements derived from dissecting monkeys and other animals. Comparison with the drawings in the *Fabrica* and *Epitome* serves to show the advance in human anatomy made by Vesalius whilst at Padua. In the course of four and a half years and before he had completed his twenty-eighth year he had finished his great work. The *De Humani Corporis Fabrica* is

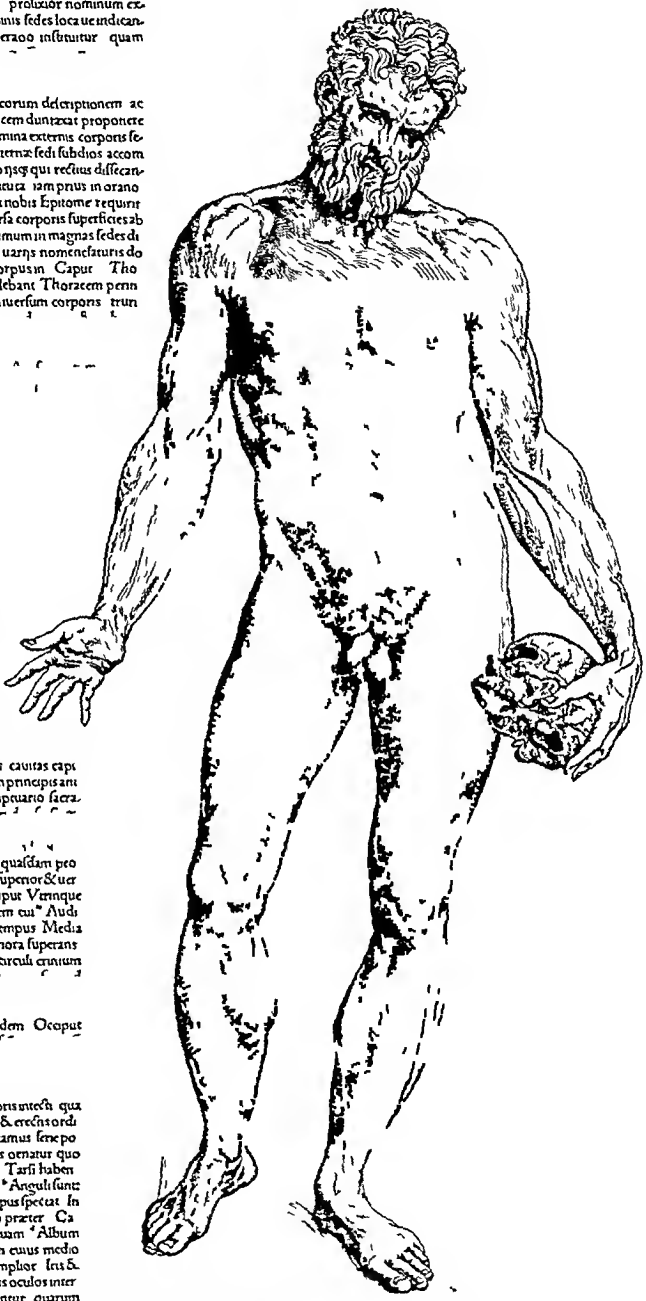
EXTERNARVM HVMANI CORPORIS SEDIUM PARTIVMVE



IC NON prolixior nomen ex-
ternas hominis sedes loca ue indicen-
tum enumeratio infertur quam

lucantiam eorum descriptionem ac
velut presentium figurarum indicem duntaxat proponere
nihil obstat quum eadem fere nomina externis corporis se-
dibus ac ossibus partibus que externae sedi subditi accom-
modentur quorum praecipua ab his qui rectius dissecan-
di rationem aggressi fuerunt influenta iam prius in oratio-
nis contextu quantum propolita nobis Epitome requirit
recensuimus. Solet itaque uniuersa corporis superficies ab
illis nominum insinuatoribus primum in magnas sedes di-
uidi ac dein illarum partes rursus uarijs nomenclaturis do-
nari. Atque ita Aegyptij medici corpus in Caput Tho-
racem Manus & Crura diuidebant Thoracem penn-
deae Aristoteles nomenclantes uniuersum corpus trun-

poris superficiem discernunt ue-
rum fides quam illi corporis trun-
cum in duas sedes primum distin-
guentes manus & crura unus par-
tis loco prima hac diuisione em-
merit illa quae Artus proprie vo-
catis constituunt extremorum no-
mine complectitur. Ac in corpo-
ris trunco duas praecipuas locant
sedes secundum duas cauitates se-
candibus inibi obuas quarum in-
terior ab eluosi interuentu septi-
transuersi sinuata iecur naturalis
alutis uenarum sedem sanguis-
cationisq; officina ac in super hinc
subministrantia organa comple-
ctitur partibus quoque genera-
tionis famulantis parata. Super-
rior cauitas cordi irascibilis ani-
mae formis uitalisq; spiritus fontis
illisq; subservientibus organis af-
scribitur. Exterior tercia corporis cauitas capi-
tribuitur cerebroque potissimum principis ani-
mae sedi animalis que spiritus promptuario sacra-



sita ac in quibus nuda lines que qualdam pro-
ponens. Frons nominetur. Haec superior & uer-
sus caput medium uertens. Sinus autem Venterque
ad sinistram latum supra que. Aurem cui. Audi-
torius meatus inest consistens. Tempus Media
capitis sedes sinistram uersus postiora superans
Vertex qui uelut centrum est. Circuli cranium

Indines dicuntur elastissimam sedem. Occiput

Oculi inferius & superius. Palpebris interfecti qua-
rum sedes ubi inuicem conueniunt & erecti ordi-
natae que ut in nauibus remotis spectamus lenepo-
sitis pilis quos Cilium nuncupamus ornatur quo-
dammodo que cartilagineae sunt. Tarsi habent
Commissiones huius termini. Anguli sunt
quorum maior nalis minor tempus spectat. In-
d f un tarum palpebrarum medio praeter Ca-
runculam in maiori angulo conspicuam. Album
candidum ut oculorum apparet in eius medio
duo se offerunt circuli quorum amplior Iris &
Coronae est minor Pupilla. Nasus oculos inter
inter cu us foramen. Nares uocantur quarum
externalis nalis. Pinnulis seu Alis uocantur. Interioris nalis constituntur. Sedes ad nalis latera alium modum pro-
munile

CITRA DISSECTIONEM OCCVRENTIVM APPELLATIONES



CENTIVM APPELLATIONES

multitracubentes Malar & quibusdam Genzuo canitur Sedes in p. 101
ter naliq totam oculorum sedema nonnullis Concus dicitur quo nomi p. 102
runt Faciepari quoniam infamius Buccae tota uero ipsius pars a su p. 103
perolys ad elanorem ulque denum emend pertinet Supra noma p. 104
xilla nonnauat reliqua autem que muraus Barba decoratur Infe p. 105
nub cuius auctus extremum Mentum nonniquam fura omni p. 106
nub edior fub Labi inferiori eubore confistens Flauor omni p. 107
fedes ad fubidit Sultuolus donata Muxla cenetur Quod la p. 108
bret anteferitur & conuenit Muxla cenetur Flauor omni p. 109
Gargareo Dentes Gingiue internat Quobiane Lingua Palati p. 110
Quod caput ad clauicula ulq ad thoracem exopt Collum & Ctr p. 111
uxit & fofterius nomen magis pofteriori parti accomodetur p. 112
und & anteriori nomen magis pofteriori parti accomodetur p. 113
genibus occurrunt Guttae acria & pofitum ipsius caput can p. 114
rabatur brachio ipsius collum legimus Humerus ueteribus uo p. 115
ad collatae dunt thoracis laeta articulus unde & partem maxime p. 116
dicunt Quod ab illo proforum uerfus Supinum humerum p. 117
radiorum accipit proforum uerfus Supinum humerum p. 118
dignior accipit proforum uerfus Supinum humerum p. 119
uicis Axilla aut Ala appellata & mufculus quos Tendines permittit p. 120
flexum producit Brachium & Launorum quibusdam Humerus p. 121
dione Pofterior flexus illius fedes Gibberus effi Patib hoc dicitur p. 122
terminum articulum dicit Cubitus & Launorum quibusdam Bra p. 123
chium & Vina ad cubitum extremum Summa manus inape quae p. 124
pars cubito ad quatuor digitorum radices porrecta in duas fedes par p. 125
atur & cubito propinquior Brachiale effi alia Polibrachia quae a p. 126
conftitutions fpecie cum pectore etiam Pectus a quibusdam Palma p. 127
nuncupatur Humeri interior fedes ravae uarq monneclus fepia mul p. 128
naq lines interfundit Volam effi alia Reliquis fumme p. 129
manus pars Digno sunt finguli tenus paribus tan p. 130
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quibus pars Digno sunt finguli tenus paribus tan p. 200

FIGURAE

de hoc amungibus etiam sequuntur Quamquam de integro
filiis etiam: Contra uero per totum Tarsis nuncupetur

a work of 759 folio pages with more than 320 wood block engravings, all arranged to correspond with the course of anatomy as given by Vesalius. It contains an account of human anatomy and physiology based essentially on the writings of Galen, the final chapter including a condensed description of Galen's experiments on animals, with a repetition of which Vesalius ended his course. The term '*fabrica*' had been used by Cicero in his *De Natura Deorum* several times, meaning 'framework', 'fashioning', 'workmanship'. Vesalius claimed that he had corrected 200 of the errors which Galen had

imported from animal anatomy. It is needless to say that he left a great number uncorrected. But there is a new edition of Galen's writings in course of publication, meanwhile, what Galen actually wrote on various points, and the interpretation to be placed on certain passages, may be regarded with a suspension of judgement.

The origin of the illustrations in the *Fabrica* and *Epitome* has been discussed at great length, the only conclusions which are at all certain are that all are original and all must have been designed by Vesalius himself. Even his opponents, Jacobus Sylvius, Colombo, Fallopio, whilst calumniating him as bitter enemies in relation to his variations from Galen, gave no hint whatever as to any copying either of illustrations or of text. Discussion has failed to establish any direct influence of the great artists who preceded him. The recent publication of Leonardo's drawings fails to suggest any relation with those by Vesalius. Leonardo for the most part dissected old people, one of his more finished drawings, viz., the lateral view of the spine, shows tipping of the bodies of the vertebra and thinning of the intervertebral discs, changes which Vesalius noted as productive of senile kyphosis. Vesalius made it his particular aim to take his illustrations from well-nourished young adults. He

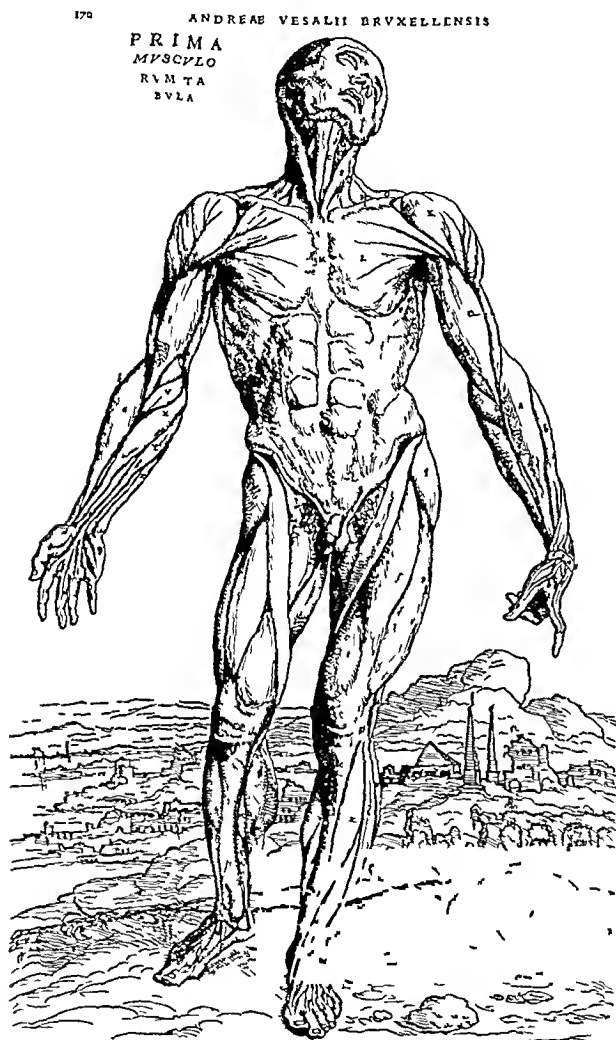


FIG. 306

quoted from Celsus the *corpus quadratum*, 'square built'—neither spare nor fat, as being fittest for illustrative purposes. Executions not only of young men, but also of young women, were frequent and thus he was able to obtain muscular bodies for dissection.

As regards the illustrations, some are well finished, some incompletely so, and a great number are schematic figures or diagrams primarily intended to explain statements in the text, statements in some cases made by Galen which Vesalius then proceeded to correct. The closest relation exists between the text of the *Fabrica* and the illustrations,

and both must be always considered in relation to the dissected preparation upon which they were based. The whole text and illustrations are linked together by an elaborate scheme of cross-references, together with thousands of marginal notes, differing according as they are printed in the inner or outer margins. The more thoroughly such points are reviewed, the more irresistible becomes the conclusion that all the illustrations must have been designed by Vesalius himself.

As to the artists who made either the preliminary sketches or the actual drawings on the wood blocks, there were then in Italy a great number of artists of the second rank assist great masters. Vesalius did not say who were the draughtsmen or who the wood engravers. Indeed he only mentioned by name two artists, and that incidentally without definitely attributing anything to them, Van Calen and Nicolo Stupio. Jan Stephanus Calcarensis (1490-1546), a native of Cleve in the Duchy of Cleves, was at Venice in 1536 where he perfected himself in the style and manner of Titian and Raphael so as to deceive even experienced critics. Whatever part he took in association with Vesalius lay between 1536 and 1539, after which he went to Naples where he was engaged in printing until his death in 1546. In the dedication of the *Tabula Anatomica* Vesalius speaks of him as a distinguished printer of the day.

In a footnote appended to the three skeleton figures it is stated that they had been printed at Venice by B. Vitalis a Venetian at the expense of Joannes Stephani Calcarensis and were on sale at the shop of D. Bernardus. In the *Epistola de cura secunda* p. 66 Vesalius said provided that he (Vesalius) could get anatomical specimens and that van Calen would not disappoint him is to drawings concerning which all that need be said about the numerous conjectures is that they are mutually destructive.

Vesalius did say that the preparation of the dissections and the direction of the eye, hand and intelligence of the artists had cost him a monstrous amount of labour. He complained bitterly of the more than wily and evasive of both draughtsmen and wood



engravers. He regretted that he had had to pay such large sums to induce skilled artists, more interested in painting Venuses and Graces, to draw pictures of skinned and foul-smelling bodies. The drawings and engravings were carried out at Padua and Venice. Vesalius watched over their preparation, at Venice he arranged the artist's proofs between the sheets of the text, before their despatch to Basel, in order to prevent mistakes in the arrangement by the printers. He himself followed and spent the best part of a year at Basel superintending the printing.

194

ANDREAE VESALII BRUXELLENSIS

QVINTA
MYSCVLO
RVM TABV
LA.



FIG 308

Underneath is a mask such as Greek actors used, for Vesalius would have the structure of the human body widely known. Vesalius is standing on the right side of a table upon which is lying a female cadaver with the abdominal viscera exposed. He is holding up his left index finger to require attention, whilst without a book he teaches anatomy directly from the dissected part. A reader, seated at a high desk, and expounding a passage from a classical author, has been superseded. Vesalius in his right hand holds a rod with which to demonstrate the object he is referring to, for he has done away

The Frontispiece (Fig 301)

—Vesalius, in the frontispiece which constitutes the title-page both of the *Fabrica* and the *Eptome*, gave a pictorial representation of his anatomical instruction. He is depicted conducting a Public Anatomy in a covered hall, lighted from above, with an apse and gallery, the architecture reminiscent, on a small scale, of St Peter's. For anatomy was a subject worthy of presentation in a noble apartment accommodating a large and general audience, rather than in a private room to a few doctors, or worse still in a mortuary cellar, temporary shed, or even in the open air. It is recorded in the archives of the University of Padua that an audience numbering 500 attended from the beginning to the end of his demonstrations.

High up in front of the centre of the apse is the coat of arms of Vesalius enclosed in a crown of laurel, three weasels, one under the other, indicating his descent from landed gentry and tracing the derivation of his name from the place Wesel, *Wesel* or *wesele* is the Flemish for weasel, and Vesalius spoke of *meæ mustelæ*. The coat of arms is supported by cupids, representing the medical students as in the Vignettes who are graving sidelong at prostrate satyrs, like gargoyles, his enemy entities.

with a separate 'ostensor' attendant behind him being engaged in keeping his knives sharp. Whilst he thus encouraged personal dissection, he made scornful allusion to physicians too delicate to handle anatomical material. At the head of the table is an articulated skeleton, and under the table are separate bones on a tray. The audience is a mixed one: there are old bearded men in robes and sandals, representing philosophers, followers of Aristotle who had taught whilst walking round the Lyceum Gardens in Athens, their need was to acquit themselves with human anatomy in place of the anatomy of animals found in the writings of Aristotle and Galen. Wealthy townsmen supported them is holding up before his eye a biconcave lens, a very early representation of the use of such in aid to distant vision. Peeping round a pillar is a fashionably dressed youth, in a slashed hose. There are nuns, one in the foreground being nudged by a bystander, the other keeping more in the background, also there are monks close behind Vesalius, Vesalius would imply that these had come to the Anatomy out of curiosity about the female genital organs, he had more than one girl at the interest the ecclesiastical clergy took in the subject of generation, theologians talked much more on this subject and its details than did medical men. Peeping round in opposite pillar is the nude figure of a well-developed young man from which Vesalius demonstrated the contours on the surface produced by the muscles, whether in movement or at rest. In the foreground the chattering tailed monkey calls to mind Galen's anatomical descriptions and the dog on a leash the animal experiments with which the course was completed. Before the table, seated on the ground are two gravediggers arguing over the division of the money received for providing the cadaver: a subject further illustrated in the Vignettes.

DE HUMANI CORPORIS FABRICA LIBER II

157
SEXTA
MYSCIPLO
FVM TA
BULA



The Portrait (Fig. 302) — The portrait of Vesalius is reproduced in both the *Fabrica* and *Uptoma* and the same block was evidently used three years later for the reproduction in the *Uptoma de radiis clupa* by which time it had become much worn. It, together

with the figure in the frontispiece, are the only two authentic representations of Vesalius. It is dated 1542, he being then aged 28. There is no signature or other mark of the artist, and there exists no authoritative statement on this point. The only thing worthy of note is that the frontispiece figure differs from the portrait, and in the second edition of the *Fabrica* in 1555 an attempt seems to have been made to alter it to an appearance more like that in the portrait. The portrait is so characteristic that it is impossible to mistake it for that of anyone else. It may, however, be a question how far the artist has deviated

towards caricature by drawing the tip-tilted nose, the keen eyes, the high forehead, the large head, the short arm, the pudgy hand, and tapering fingers. In the frontispiece the hands are drawn fine and well proportioned, more fitting to one skilled in handicrafts.

Vesalius is standing holding up the dissected right arm of a well-developed female. Before him is an opened page, such as he was then engaged in writing, dealing with the muscles moving the fingers. There is here a suggestion of the triple relation between the text, the illustrations, and the dissected part. It was a region which Galen had treated superficially, owing to its small size in the monkey.

On the side of the table is the motto "*Ocyus jucunde tuto*." Celsus, quoting from Asklepiades, wrote '*ut tuto, ut celeriter, ut jucunde curet*' (to treat with safety speedily, and pleasantly). Vesalius applied the quotation to his teaching of anatomy but the variation from '*celeriter*' to the latinized form of the Greek *ὀξύς* may be based on a tradition different from that through Celsus.

The Vignettes enclosing the Capitals—In the vignettes Vesalius included a remarkable series of thumbnail sketches, which, whilst superficially they form



FIG. 310

humorous skits on the medical students, portrayed as fat naked boys, cupids, and moretti, are of great historical interest because they indicate the difficulties attending the pursuit of anatomy, and incidentally enforce the importance of personal dissection of human cadavers as well as of animals.

The large initial Q of *Quantumvis* ('however much soever') which commences the preface of the *Fabrica* dedicated to Charles V, depicts the vivisection of a young pig, one cupid is reading out of a book, whilst another is cutting into the animal's neck in order

to repeat one or other of Galen's experiments either the division of the vagi nerves to show then control of the voice, or the performance of tracheotomy and the examination of the mechanism of breathing.

The vignette enclosing the huge capital 'V' shows cupids disinterring by candlelight a stout cadaver, one cupid dressed out in imitation of a Roman soldier with helmet and spear, is on guard as a sentinel, another serving as a scout is running up holding a flag, to give an alarm. The large O shows cupids macerating a skull by boiling in a iron pot hanging over a wood fire. Vignettes enclosing smaller capitals represent cupids catheterizing one of their number, disinterring and breaking open a coffin, sawing open a skull, putting a fractured leg into a glossocomium or box splint, applying the cantery over the temporal artery through holes in a plate. There are also sketches of cupids performing paracentesis abdominis on an old man, taking down from the gallows a female cadaver, inflating a stomach to demonstrate the fibrous coat, carrying off in procession for triumph steps a cadaver for dissection, taking down from the top of a pole a decapitated head, studying an articulated skeleton, examining the abdominal viscera, removing the eye from the head of an ox, and performing venesection. These sketches are not scattered arbitrarily, but are related to the text which follows the respective capitals.

In the vignette of the large 'V' forming the initial letter in the preface of the second edition there is a hit at his critics. The Phrygian Mursyas, who had ventured with his flute to match himself against Apollo and his lyre, is being skinned alive, whilst two nymphs look on. At the back of the *Fabrica* Arion with his lyre, after being cast overboard is riding on the dolphin's back. This is varied in the second edition, where it is represented that the dolphin has brought Arion safely to land.

Instruments used in Dissecting—Some of these are seen on the table in the frontispiece. Vesalius exhibits in various passages an extensive acquaintance with handicrafts of various kinds. One chapter is devoted to the instruments employed in dissecting and some thirty are drawn lying on a table (*Fig 303*) knives like razors or

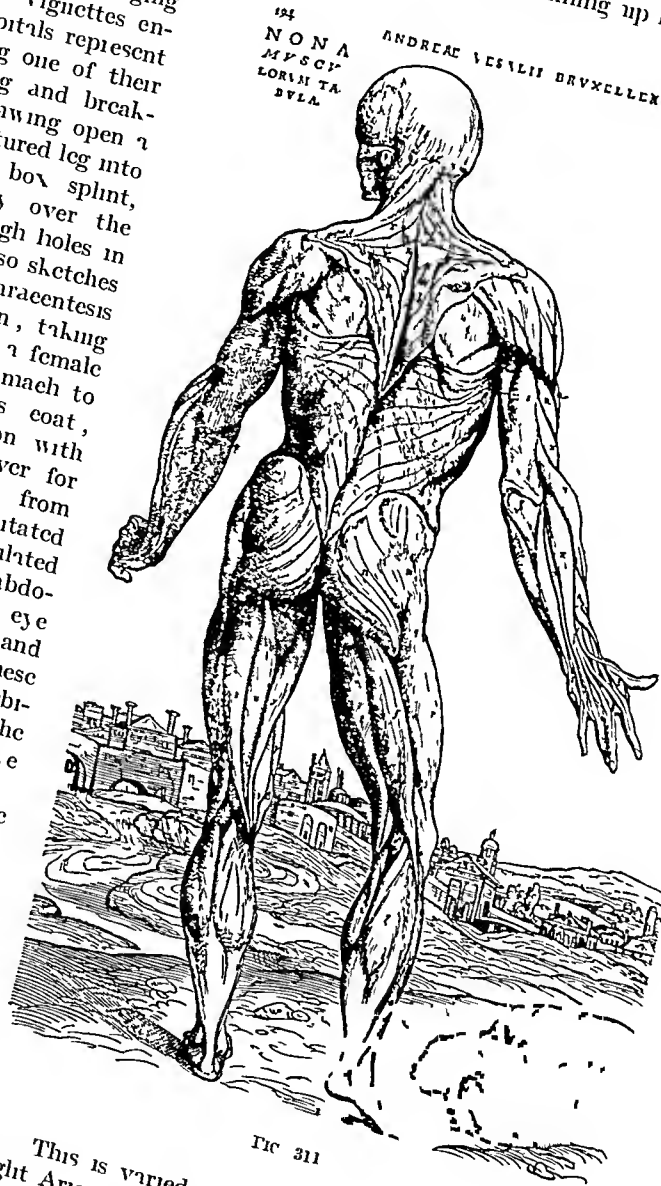


FIG 311

dinner knives, penknives, also knives shaped out of boxwood and ebony for dummy operations, two-pronged sharp hook retractors, probes and probe-pointed ductors and cannulae, needles, strong curved chisel ended as used in bookbinding also smaller triangular and spear-ended as used in surgery, a strong butcher's saw, scissors in shape like trowsers', sharp-pointed, a wooden mallet, hollow reeds for inflating the lungs and other organs, brass wire for articulating skeletons, along with awls of various sizes fitting into a common handle, pincers for twisting up and nippers for cutting the wire. There are no anatomy

forceps, Vesalius used his fingers, including his finger-nails, chain hooks were a later invention¹

206
DECIMA
TERTIA
TABULA

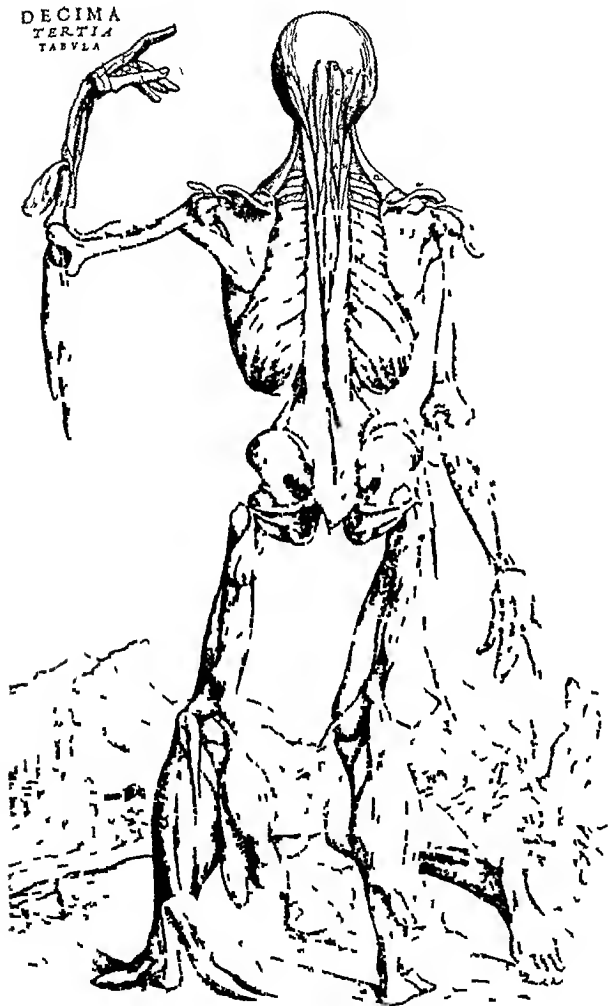


FIG 312

reduced in the *Fabrica* to one fifth of the natural height, viz., to 340 to 345 mm—i.e., one-fifth of 1700 to 1725 mm, the average height of a man. In the *Epitome* the reduction is to one-quarter, the figures measuring 425 to 430 mm in height.

The male and female nude figures (Figs 301, 305) produced in the *Epitome*, although from an artistic point of view they bear comparison with such earlier (and 1504) wood block engravings as Dürer's 'Adam and Eve', were in fact designed to conform with the other figures. The contours on the surface of the male nude exhibit the effect of the contractions of muscles in correspondence with the positions in which the head, trunk,

The Muscle and Skeleton

Figures—The illustrations designed by Vesalius for the purpose of delineating the muscles and skeleton form a remarkable series. There are in the *Fabrica* sixteen muscle and three skeleton figures, in the *Epitome* two nude figures and five of the muscles, all drawn from well-developed young adults. It is a special mark of the genius of Vesalius that he succeeded in portraying the muscles as if in the state of contraction required to produce the particular position given to the figure, and the skeletons suggest life and movement. The position and balancing of the head, trunk, and limbs vary with each figure, and by cross-references an interrelation between the different figures is established, so that each muscle is presented from several points of view. If we direct particular attention to movements of the arm at the shoulder-joint or to those of the leg at the knee and ankle, the various illustrations taken together serve to analyse the way in which such movements are produced. Vesalius was accustomed to demonstrate as many as forty muscles one after the other. The figures are drawn in correct proportion being

and limbs are portrayed These muscles are shown in the muscle figures after their exposure by dissection The female nude, whilst in some respects resembling Greek models, is in general designed to serve as a contrast to the male nude in respect to the positions of the head and limbs Although, as compared with the male, the surface contours are smoothed down, the shading produces the external appearance of a young adult woman But Vesalius had a further design for the figure, which he adapted for the superposing of the diagrams There are no letterings to blur the surface of these nude figures, for the width of the page in the *Epitome* permitted of a brief naming of superficial positions in a column of text printed alongside each figure Whether on his living subject, or on the cadaver before the actual commencement of dissection, Vesalius was accustomed to sketch the outline of the bones, superficial veins, etc

DE HUMANI CORPORIS FABRICA LIBER I
HUMANI COR-
SIMPL COMPACTO
EX FACIE EXPRES
PORIS OSSIVM
RVM ANTERIORI
SIO

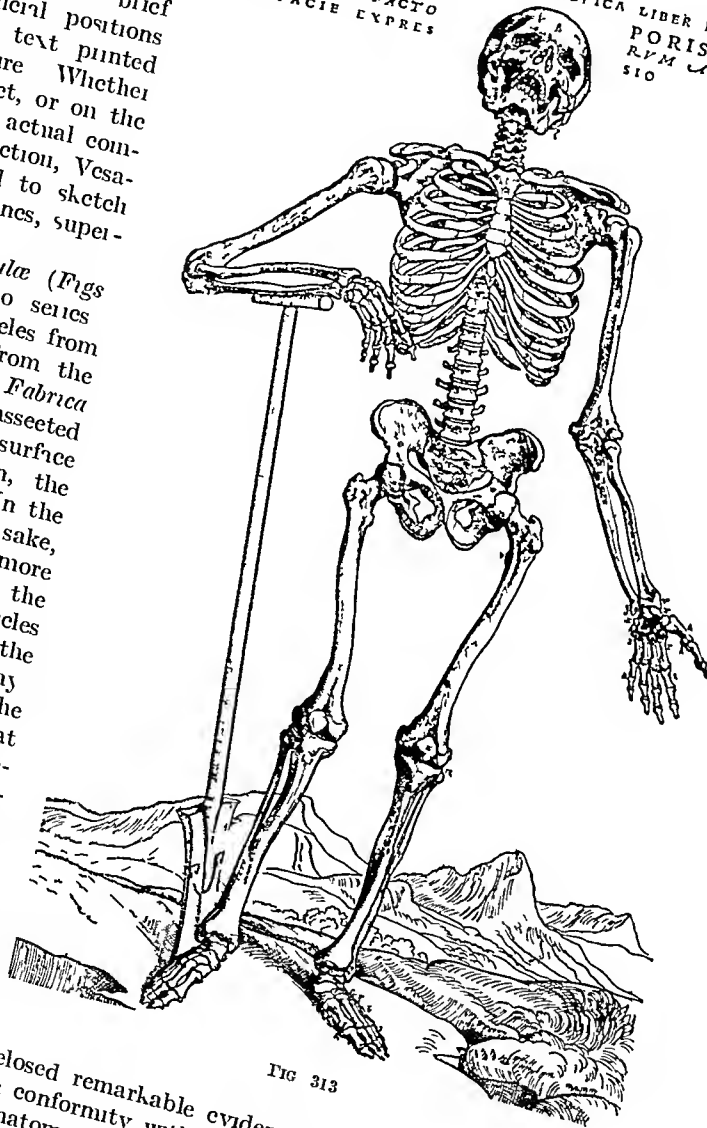


FIG 313

The muscle *Tabulae* (Figs 306-312) fall into two series the one exhibiting muscles from the front, the other from the side and back In the *Fabrica* the muscles are drawn, dissected in four layers from the surface inwards to the skeleton, the same on the two sides In the *Epitome*, for economy's sake, the right half exhibits the more superficial layer of muscles, the left half the layer of muscles exposed when those seen on the right side have been cut away The result as regards both the *Fabrica* and the *Epitome* is that every muscle comes to be depicted several times, each muscle is drawn, not only in position, but also raised from its origin, by which plan the belly of the muscle is exhibited, contracted is when in action, hanging dependent from its tendon of insertion

If the figures are submitted to a close examination, there is disclosed remarkable evidence of ingenuity in designing the position of the general figures in conformity with the text Taking them generally, the figures are free from animal anatomy But Vesalius, both in the text, and by repetition in the indexes of the illustrations, laid emphasis on corrections he made to Galen's descriptions Instead of describing the separate flexor and extensor muscles of the thumb and great toe, Galen had noted merely slips from the tendons common to the fingers and small toes The description of the plantars and of its insertion into the

os calcis to the inner side of the tendo Achillis collected Galen's statement, derived from the monkey, that the tendon of the plantaris was continued into the plantar fascia. Galen had described the popliteus muscle of the monkey as the chief flexor of the leg upon the knee, whereas Vesalius showed it to be in man a small and relatively unimportant muscle.

In two instances in particular, however, Vesalius introduced confusion, which separate figures would have avoided. In order to illustrate Galen's description of the scalene and

the rectus abdominis in the dog and monkey respectively, he added to the human muscle a drawing of the extension of this muscle in animals. Whilst in other figures the human rectus abdominis is depicted, in the 5th muscle *Tabula* (Fig. 308) of the *Fabrica* there is drawn a continuation of the muscle upwards over the front of the ribs, as far up as the first rib, special lettering and a dividing line mark this extension of the rectus abdominis found in long tailed monkeys.

In several figures the human scalene, triangular in shape, is treated as an undivided muscle. In the 6th muscle *Tabula* (Fig. 309) in order to illustrate Galen's description drawn from the dog, the muscle is shown continued as a strip over the front of the ribs, anterior to the serratus magnus, the extension being likewise distinguished by special lettering and lines.

The following are noteworthy instances of detail in the drawings. In the 5th muscle *Tabula* (Fig. 308) the pyramidules are distinguished from the recti abdominis muscles, by examining the series in which the external and internal oblique and the transversalis abdominis muscles are portrayed, the course of the spermatic cord through the internal and external ring is demonstrated. In the 7th muscle *Tabula* (Fig. 310) the under sur-



FIG. 314

face of the diaphragm is in view whilst in a separate figure the detached diaphragm is exhibited with its crura, central tendon and peripheral muscle. Vesalius, who was fertile in his comparisons, likened the shape of the detached diaphragm to a sting ray but added that it was not a very good comparison. He held that Galen had described the oesophagus as passing behind, whereas he drew the opening for the passage through the diaphragm of the oesophagus on the left, and that for the ventrals on the right.

The representation of the muscles from the side (Fig 307) and back (Fig 311) show the superficial muscles in action, and are valuable illustrations from the artistic point of view. The deeper layers of the muscles (Fig 312) were of importance, said Vesalius, because it was by them that man gained a superiority over animals as regards the mobility of the spine, and the free movements of the shoulders, hip, and other joints. In animals the spine was rigid, and the shoulder and hip movements were limited to one movement backwards and forwards.

DE HUMANI CORPORIS FABRICA LIBER I
CORPORIS
POSTERIORI
HUMANI OSSA
FACIE PROPOSITA

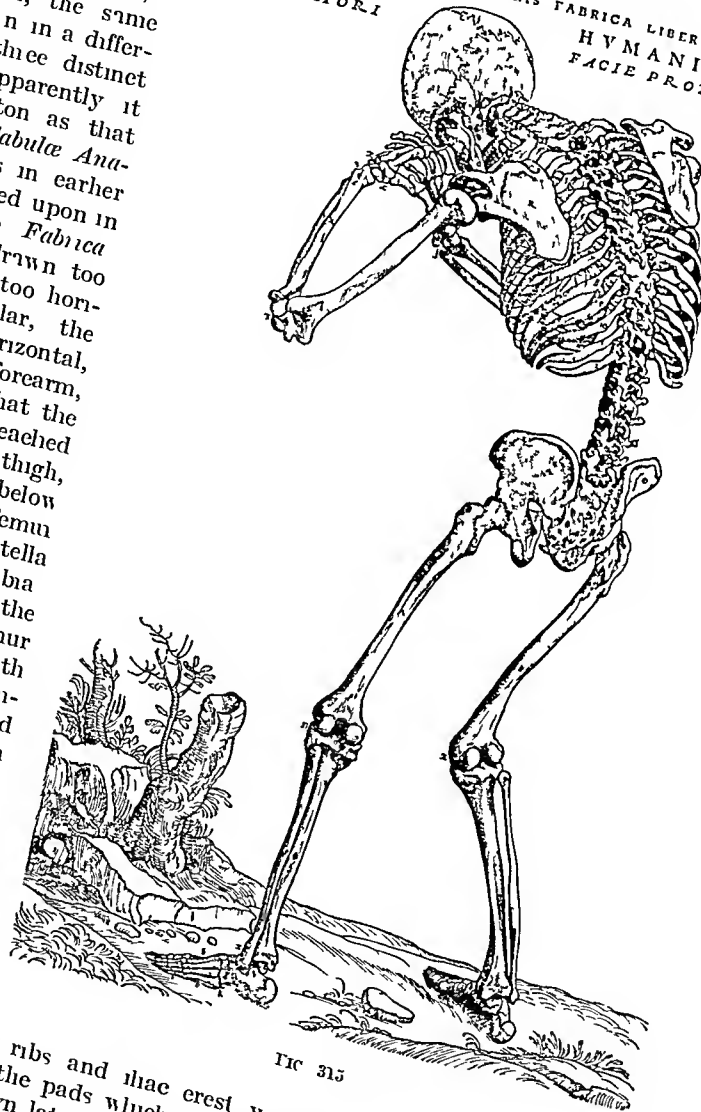


FIG 315

The three *skeleton figures* at the end of the first book of the *Fabrica* were drawn from the articulated skeleton of a young man, age 18, so that the epiphyseal lines are shown, the same position from three distinct points of view. Apparently it was the same skeleton as that represented in the *Tabula Anatomica*, if so, defects in earlier drawings were improved upon in those included in the *Fabrica*. The skull had been drawn too flask-shaped, the pelvis too horizontal and quadrangular, the neck of the femur too horizontal, the upper limb, arm, forearm, and hand over long, so that the tip of the middle finger reached and the wrist-joint much below the great trochanter, the femur too long for the tibia, the patella overlapping the head of the tibia. In the *Fabrica* the lengths of the humerus and ulna, of the femur and tibia, agree fairly well with the data in present day anatomical works, the pelvis is tilted forwards, the neck of the femur set at an obtuse angle to the shaft, the patella placed between the condyles of the femur. Even so, Vesalius himself criticized some figures, saying that the thorax had been articulated so as to make it too barrel shaped, the upper edge of the ribs had not been turned inwards enough, and the distance between the lower ribs and iliac crest was exaggerated, apparently owing to the excessive thickness of the pads which represent the intervertebral discs. Indeed, his figures of the thorax drawn later show a remedying of these defects. The first skeleton figure (Fig 313), with the right elbow resting on the gravedigger's shovel, exhibits the epiphyseal lines of the acromion, head of the humerus, lower end of the radius, lower end of the femur, and upper and lower ends of the tibia. Among other details there is drawn the radio-ulnar triangular cartilage, elsewhere Vesalius

CHARTA EA QVA FIGVRAM PARARE CONVENIT ILLI QVAL
NERVORVM SERIEM EXPRIMIT APPENDENDVM

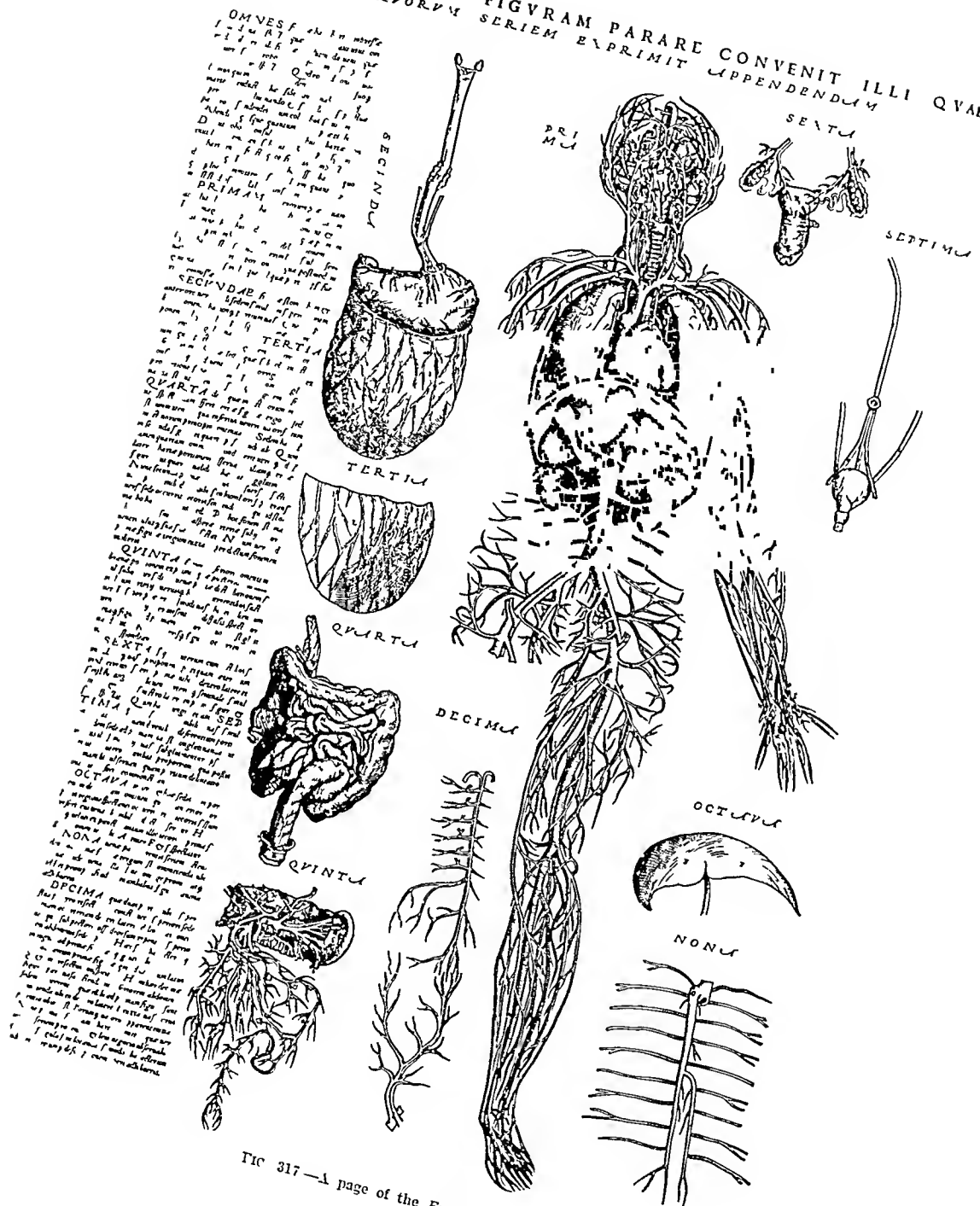


Fig 317—A page of the Fpitoeme

described and drew the interarticular cartilages of the knee, of the temporomandibular joint, and of those at each end of the clavicle. The sesamoid bones are shown, included with them being the pisiform bone, and the os vesalianum separated from the projection at the base of the fifth metatarsal.

The second skeleton figure (*Fig 314*) leaning on a monument, has supplementary figures in order to show the under surface of the skull, the hyoid bone, and the malleus and incus, lying on the top of the monument. This was the figure repeated in the *Epitome*, because such is the position of the skeleton that practically all the bones and joints are exhibited, and that on both aspects front and back, with one or two exceptions—the sacrum and coccyx and the lesser trochanter of the femur cannot be seen.

The epitaph on the monument has for its aim the countering of the objection to the examination of the body after death. *Vivitur ingenio cetera mortis erunt* ('Man's spirit lives all else death's hand shall claim' *English by Miss Joyce Love, M.A.*)

The third skeleton figure (*Fig 315*) is drawn from a standpoint half-way behind and to the left, in a position in which the spine is bowed forwards with the forehead resting on the clasped hands. Thus the skull, spine, scapulæ, and shoulder joints are viewed from a peculiar position. By close examination of the three figures the complicated interrelation in the several designs can be appreciated, thus the shoulder and hip joint, the upper and lower limbs, are portrayed each in half a dozen different ways, varying with the several positions. On the other hand, each of the three figures supplies some representation not in the others. In the third figure the sacrum and coccyx are shown in their human curve without any suggestion of a tail, the sacrum is represented as formed by six bones, because so Vesalius said, that was the case in the particular skeleton drawn.

Now supposing we direct our attention to movements with which we are familiar, such as overhand bowling at cricket or serving at tennis, in which the arms have the major rôle, or to those in running and jumping, in which the legs play the chief part but in which the simultaneous movements of the arms are of importance, or, thirdly, to rhythmic movements such as the performance on the stage of the dance of the Greek warrior—all of which complicated motions may now be viewed by means of photography on the moving films, and the movement of joints through an x-ray screen—it is remarkable evidence of the genius of Vesalius that his muscle and skeleton figures may be used in the analysis of these and other complicated movements.²

Horizontal Sections through the Brain—The illustrations of the human brain in the *Fabrica* are noteworthy, because on examination it becomes obvious that the drawings were made directly from the anatomical preparations. Not only are there drawn parts which are named, but there are outlines which, although unnamed, can be recognized as representing structures which the draughtsman had under his eye.

Figure 4, page 608, is a drawing of a horizontal section through a man's brain which had been made at the level of the corpus callosum. The part of the left hemisphere thus cut off was turned over so that in a separate figure the roof of the lateral ventricle formed by the lateral expansion of the corpus callosum could be depicted. At the margins of the hemispheres the outlines of the convolutions and sulci are marked, that external to the undulating lines being stated to be yellowish or greyish owing to the supply of blood through the vessels of the pia mater, and that internal to the waving line white, dotted with red points. In front and behind the lateral ventricles and between the hemispheres lay the corpus callosum, distinguishable from the rest of the brain. Between the ventricles the hinder limb of the fornix is drawn, although not named in this figure. The floor of the lateral ventricle on each side exhibits outlines of the nucleus caudatus of the corpus striatum, and also of the optic thalamus, overlying this is seen the choroid plexus receiving cobweb-like veins and turning down into the descending horn. The tentorium thalami and the stria medullaris are drawn, but not named.

Figure 5, page 609, the next figure, represents a horizontal section immediately under the previous one. The corpus callosum is drawn after being divided in front at its genu,

raised and turned back so that its posterior limbs, coming up from the descending horns, are seen to unite to form the body. The tapering off in front shows the junction of the body with the anterior limbs. A ridge along the under surface of the corpus callosum corresponds with a groove on the dorsum of the fornix. This marks the septum lucidum, which Vesalius said could be best demonstrated by looking at it sideways whilst the corpus callosum was held up with the fingers of the two hands. To give one instance of his fertility in comparison he said it resembled a precious stone ground thin, a sheet of mica set in a window or door, or the wafer used at Mass.

Figure 6 page 610, shows the fornix raised and turned back from the front, thus exposing the meeting of the choroid plexus of each side, which then was reflected as the tela choroidea or velum interpositum, to become continuous with the vena magna Galeni, and through that with the straight sinus and the torcular Herophili. The choroid artery from the internal carotid is shown entering the extremity of each descending horn to join the end of the choroid plexus. A groove between the two optic thalami, forming the middle line of the third ventricle, leads forwards to the infundibulum and backwards to the aqueductus Sylvii.

Figure 7 page 611, depicts a horizontal section below the floor of the lateral ventricles. There is thus shown in horizontal section the optic thalamus, the nucleus caudatus, the nucleus lenticularis and—between them—the internal capsule. Outside the nuclei is drawn the external capsule and further out still an outline of the island of Reil.

Thus and the following figure afford a peculiar proof that the artist had before him the anatomical preparation, and drew what he saw. On the right there are lines indicating the division of the lenticular nucleus into the putamen and the globus pallidus, on the left the horizontal section has dipped a little lower so that there appear lines indicating commissural fibres between the globus pallidus and the optic thalamus, although no name is given to these lines.

Figure 8, page 613, also shows the left side cut a little lower than the right, which not only confirms the above, but affords the additional evidence that the sections were made in series from the same brain.

The Nerve and Vein and Artery Diagrams—The diagrams which Vesalius inserted in the *Fabrica* and *Epitome* should be considered after taking due note of the explanations concerning them included in the text. Critics have merely glanced at them in a superficial way, and condemned them as rough, untrue, or antiquated. The nerve diagram (Fig. 316) was drawn for the *Epitome*, indeed it may have formed the last drawing for the *Tabulae Anatomicae* which is missing from the Collections reproduced by Stirling Maxwell and by Holl and Sudhoff. Being drawn for the *Epitome* and so of greater length, when bound up in the *Fabrica* the lower fourth had to be infolded. Explanations concerning this diagram are given (*Fabrica*, 1543, iv, pp. 353, 354, p. 338, 86, i, line 14, and 1555, iv 532, 87, 1). The same remarks apply to the combined vein and artery diagram (Fig. 317) (*Fabrica*, 1543, iii, p. 313).

These diagrams occupy the full length of the pages of the *Epitome*, where it is stated that for elementary instruction they were intended to be cut out and then applied over the female nude and other full-length figures.

The Museum of the Royal College of Surgeons possesses the *Tabulae Evelynae*, the origin of which was described by Evelyn in his *Diary*. On comparison with the nerve and the combined vein and artery diagrams in the *Fabrica* and *Epitome*, the relationship of the latter with the *Tabulae Evelynae* becomes apparent. Before the introduction of methods for preserving anatomical material from putrefaction, and for the injecting of blood-vessels, nerves and blood-vessels were rapidly dissected out and then spread on boards for inspection and drawing, after being varnished over they could be preserved. Evelyn's account shows that, a century after Vesalius, his successor in the Chair of Anatomy at Padua, Vesling supervised the preparation for Evelyn of human nerves and blood-vessels by the same method as had been adopted by Vesalius when preparing his diagrams.

The examples I have referred to explain why the illustrations designed by Vesalius form an important foundation upon which the present knowledge of human anatomy is grounded. But the general impression gained by a study of the writings of Vesalius leads further to the conclusion that he had a wider aim than that of instructing students of medicine and of art. He would have an educated man possess a knowledge of anatomy and physiology, and there was a tendency in this direction during the 16th and 17th centuries. Christopher Wren and Evelyn may be cited as studying anatomy seriously, although perchance others, like Pepys, may have gone to an Anatomy at Barbers' Hall out of curiosity.

To day, outside the wide boundaries of medicine and of art, few can be said to follow the exhortation implied in the letter by Vesalius dedicating the *Epitome* to Prince Philip —

"You will think it base and unworthy that, while such varied courses of study are pursued, the composition of the body which accompanies us through life should be a secret from us, that man should be absolutely unknown to himself, and that we do not examine the construction of the organs formed so perfectly by the Almighty Designer of the Universe. The vital activities of these organs by which everything is accomplished we confine ourselves merely to wondering at."

I conclude with the following epigram by Vesalius —

I am good health, put to the test and tried,
Unhappy mortals I am here to guide,
Unless indeed I'm snatched off by some blow,
Or play the run-away before some foe
At first, in very truth, a tempest black
Of jealousy and envy held me back,
For few essays there are so I have heard,
That fear of envy hath not first deterred
But yet at last, despite Sir Envy's sway,
Despite all jealousy, I broke away,
I burst my bonds, to none would I be thrall
My name is known I shall be read by all
Do only thou, dear Reader take in hand
This fruitful work and spread it through the land,
With cheerful brow assist me in my task
And make the dutiful attempt I ask
Foster the strength that grows nor treat with scorn
The strength that deep within the soul is born,
For 'tis that strength which in the after days
Shall bring forth fruit from slight and scant essays

(English renderings by Miss Joyce Lowe M.A.)

The illustrations reproduced in this paper are from photographs of wood block engravings in the *Fabrica*, 1543, and from the copy of the *Epitome* printed on vellum in the British Museum Library.

NOTES

¹p. 394 —The *Fabrica* was subjected to indiscriminate plagiarism (I *planarius* a kidnapper). The woodcut 303 p. 385 illustrating the apparatus used by Vesalius in dissecting with the addition of a bleeding bowl and centrebit appears in *Clowes On Gunshot Wounds* 1637 Cap. 27 to illustrate a note of certain necessary medicines and instruments good for young practitioner of surgery and thus in turn was copied by Curl as a woodcut of Clowes showing his surgical instruments (Curl *Geschichte der Chirurgie*, 1898 in 365).

²p. 400 —The drawings of an articulated skeleton from four points of view preserved in the Uffizi Gallery, Florence and attributed to Leonardo da Vinci were made whilst the skeleton was hanging loosely from a peg (See Holl and Sudhoff *Archiv für Geschichte der Medizin* 1914 vi. 334 Tafel viii).

THE EFFECT OF GASTRO-ENTEROSTOMY ON GASTRIC FUNCTION, AS INTERPRETED BY THE FRACTIONAL TEST MEAL.

By ERNEST F GUY, MANCHESTER

THE method of investigation of the functions of the stomach by the fractional test meal as introduced by Rehfuess,¹ has hitherto been applied chiefly as an aid to diagnosis. The present investigation represents an effort to study the effect on the gastric functions of the operative measures adopted in the treatment of chronic ulceration of the stomach and duodenum, and to account for the varying results obtained.

The old method of withdrawal of a single specimen one hour after a test meal is unsatisfactory, since it demonstrates but one phase in a constantly varying cycle. By employing the fractional test meal, the state of secretion and motility of the stomach can be followed throughout the course of digestion.

Patterson,² basing his views on the results obtained with the old type of test meal, found an average reduction of 30 per cent in the gastric acidity after gastro-enterostomy, and a slight acceleration or retardation of the rate of emptying, within physiological limits. On these assumptions he attributed the beneficial effects of the operation to the diminution of hyperacidity, and not to improved drainage.

The application of the fractional test meal to the problem does not appear to have received attention by many observers.

Bonar³ stated that the post-operative acid level varied with the position of the ulcer, whilst Wilensky⁴ did not find any relation between the position of the ulcer and the character or intensity of the changes. In a recent paper⁵ the latter writer appears to attribute the variations after operation to differences in operative technique.

Material employed in the investigation.—Over 50 cases have been examined, and, of these, the presence of an ulcer has been confirmed at operation in 31. These have been provisionally classified according to their position, as gastric, pyloric, and duodenal, and were distributed as follows:—

Gastric ulcer	3 cases
Pyloric „	9 „
Duodenal „	14 „

In 5 cases, whilst it is known that an ulcer was found at operation, no information has been obtained as to its exact position.

Twenty-six patients have been examined after gastro-enterostomy, and in 14 of these the curves before and after operation have been obtained. The remaining cases consist of patients presenting chronic abdominal symptoms due to causes other than ulceration. In many of these the absence of an ulcer and the presence of some other disease was demonstrated by laparotomy. The majority of the patients were pensioners receiving treatment at the Ministry of Pensions Hospital, Grangethorpe, Manchester.

Technique.—The usual method of withdrawal, by a Ryle's tube and syringe, of specimens of the gastric contents at 15 minute intervals after the swallowing of a pint of oatmeal has been employed. Certain points have been developed in the technique of the examination which are believed to have important effects on the resultant curve.

The quantity withdrawn each time is limited to 2 c.c. To obtain a larger quantity often necessitates a pressure in the syringe sufficient to cause minute hæmorrhages from the gastric mucosa. Whenever blood has been found in the specimens of the present series of test meals, subsequent laparotomy has invariably proved its source to be an active ulcer.

The examination is continued until sixteen specimens have been taken, i.e., three and three-quarter hours after the test meal, in order to obtain a record of the 'after secretion'. If large quantities of mixed food and secretion are removed, the test is often brought to an end too early by the complete emptying of the stomach.

A still more important modification is the early filtration of the gastric contents. As the specimen is withdrawn the syringe is emptied immediately on filter paper. This precaution obviates the marked changes in acidity which take place *in vitro* within the course of a few hours. A number of control titrations have now been made, from which the two following examples are taken. 'A' represents the titration figures of the specimen filtered immediately, 'B' those from the control specimen filtered after standing in the test tube for four to six hours at room temperature.

		Free HCl	Total Acidity
Example 1	A	7	16
	B	0	11
2	A	15	25
	B	7	17

The amount of difference in acidity due to chemical changes proceeding *in vitro* depends on the stage of digestion at which the specimen is taken, the greatest variations being noted when food is present. This variation is particularly marked after gastro-enterostomy, and may then result in a total disappearance of free HCl, probably accounting for many of the unacid and subacid results claimed after this operation.

The titration is performed with a burette of small calibre with $\frac{1}{100}$ c.c. graduations, and fitted with a capillary dropper. By using this method accurate titrations can be made with 1 c.c. of filtered juice, or even with less on the rare occasions when this quantity is not obtainable. The first specimen is taken before the test meal is given, and in the more recent test meals the stomach has been completely emptied at this time in order to measure the quantity of resting juice. The disappearance of starch from the stomach is taken as the simplest means of indicating the final passage of food from that organ.

Bolton and Goodheart⁶ have demonstrated the lowering of the acid curve caused by regurgitation through the pylorus of alkaline fluids even in the normal stomach, but despite this the height reached by the acid curve may be taken as a fair indication of the quantity of the total gastric secretion.

GENERAL RESULTS OF GASTRO-ENTEROSTOMY

The motor and secretory functions of the stomach are profoundly modified by the existence of the new stoma, which permits a readier exit for the food and a freer entrance for the duodenal contents. The presence of the latter in the stomach is indicated by the appearance of bile in the specimens removed. The fractional test meal shows that bile is constantly present in the stomach after gastro-enterostomy. A very occasional single specimen is free from bile, but it is invariably present in the majority of specimens obtained during the course of the test. As bile is present it must be assumed that pancreatic juice has an equally free means of entrance, though its presence cannot be detected by any simple chemical test.

The freedom with which the duodenal contents now enter the stomach was well shown in one case in which x-ray examination after a bismuth meal proved that all the food was passing through the pylorus and none through the stoma, although the latter was found to be patent and of sufficient size when the abdomen was opened at a later date. Despite the fact that the stoma was apparently functionless as regards food, bile was present in every specimen of gastric contents examined.

The quantity of resting juice found in the stomach after gastro-enterostomy is extremely variable, more so than is the case before operation. Quantities varying from 5 c.c. to 150 c.c. have been recorded. This degree of variation is probably due to the freer communication with the jejunum which now exists and in the present series the

quantity has appeared to bear no relation to the position of the ulcer. A possible fallacy in this estimation is the passage of the end of the tube through the stomach though every effort is made to avoid this by keeping the measured mark on the tube opposite the incisor teeth when the specimen is taken.

On the motor side, so long as the stoma is functioning, the rate of emptying is always considerably more rapid than normal.

The gastric acidity is lowered by the influx of alkaline fluids. Pancreatic juice is the chief neutralizing factor, since it possesses ten times the alkalinity of any of the other fluids concerned. It is conceivable that the amount of alkaline fluid entering the stomach through the stoma would, at times, be sufficient completely to overcome the acidity of the gastric contents and to render them alkaline, but this has never been found to obtain after gastro-enterostomy. In this respect the following case is instructive.

Case—J S, age 34, was admitted to hospital with a history of gastro-enterostomy performed ten months previously for symptoms of abdominal pain and vomiting which had existed for two years. These symptoms recurred very shortly after operation.

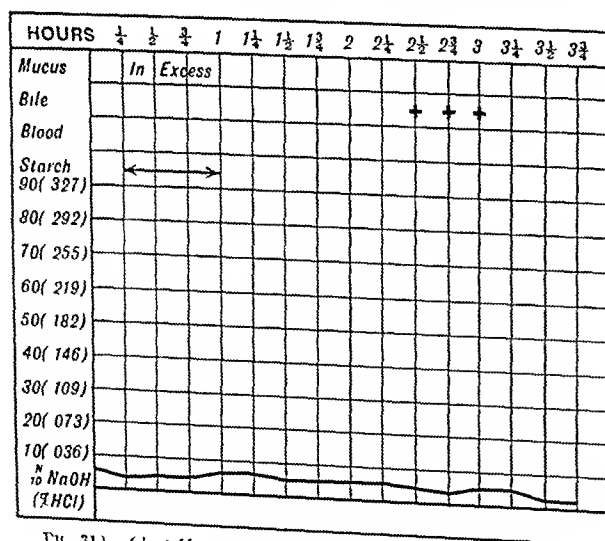


FIG. 311—Chart II. Same case as in Fig. 310 after excision of the gastro-enterostomy stoma.

tion of the gastric contents, a low degree of acidity was still maintained. The level to which the acidity of the stomach is reduced by gastro-enterostomy depends primarily on the position of the ulcer for which the operation was performed. It is therefore necessary to discuss the effects under different headings and to consider the altered state of the gastric functions previous to operation.

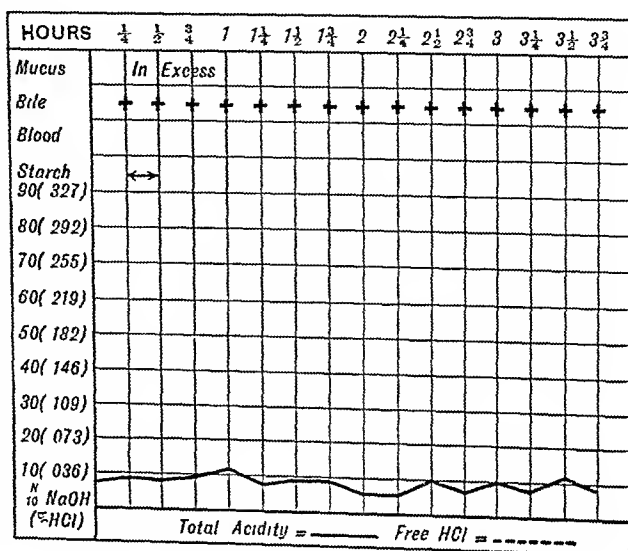


FIG. 310—Chart I. Case of achlorhydria in which gastro-enterostomy had been performed.

A fractional test meal on Nov. 26, 1921, showed a total absence of free acid and a very low total acidity, as can be seen in Chart I (Fig. 310). On Dec. 8 the abdomen was opened, and as no trace of an ulcer or of pyloric obstruction was found the gastro-enterostomy stoma was excised and the normal continuity of the gut restored.

A fractional test meal taken one month after operation and repeated on Oct. 31, 1922, gave the curve shown in Chart II (Fig. 311). The quantities of juice obtainable on each occasion were extremely small and a condition of achlorhydria was still present. Examination of the digestive power of the juice demonstrated the presence of pepsin, which could be activated by the addition of HCl.

In this case, although the conditions present after the gastro-enterostomy were such as to favour the complete neutraliza-

For purposes of comparison a normal curve will first be described

The Normal Curve—The normal curve given by Crohn and Reiss,⁷ and largely adopted as a standard, is constructed from the average of a number of readings, and does not show features which are typical of an individual curve, nor is it continued sufficiently long to show the after-secretion. Whilst it is realized that different apparently normal individuals show considerable variations in their curves, and that smaller variations may occur in the same individual at different times, the curve in *Chart III* (Fig 320) shows most of the features which may be regarded as typical of a normally functioning stomach. It is owing to these variations that the actual shape of the curve is of but little importance, and attempts to classify curves on this basis can produce no useful results.

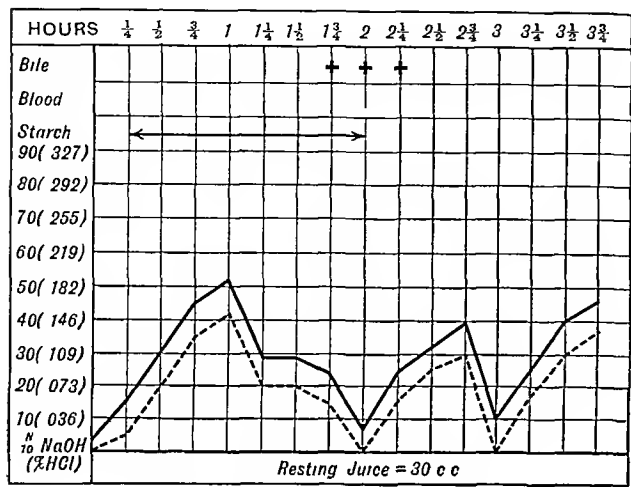


FIG 320—Chart III A typical normal curve

starch within two and a quarter hours. Longer periods than this are regarded as an indication of delay. Disappearance of starch within one and a half hours or less indicates ‘hurry’.

The normal stomach always empties on a curve of falling acidity.

2 Bile—This is constantly found in the stomach when the last portion of the food is passing out, and reappears at least once during the later stages of the test when the stomach is empty of food.

3 Resting Juice—The quantity is variable. Fowler, Rehfuess and Hawk⁸ examined over one hundred normal persons, and found an average of 52 c.c., with variations from 23 to 160 c.c. The total acidity of the normal resting juice is low, and should not rise above 20. Usually it is lower than this, and free acid is often absent. Bile is occasionally present.

4 Acidity—The acidity rises to a maximum in about one hour, the maximum being about 50 with an HCl reading of near 40. From this level the acidity falls as the stomach empties. After food has passed from the stomach the secondary rise (which may be single, or double as in the example shown) of the after secretion occurs. The acidity of this secretion reaches a level as high as, or even higher than, that observed during the course of gastric digestion. It will be noticed

The following are the features of the normal curve

1 Motor Power—The gastric contents should be free from

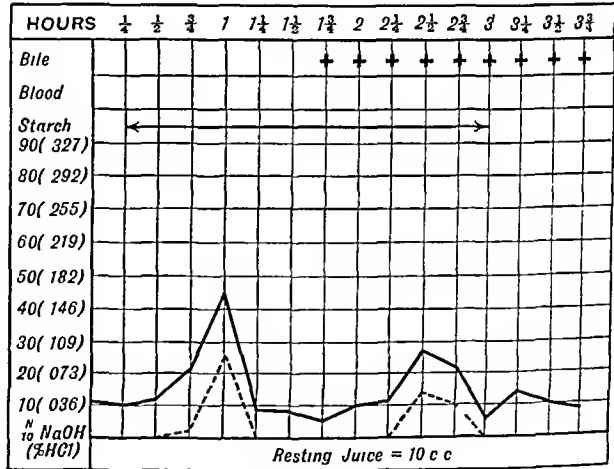


FIG 321—Chart II Gastric ulcer

that the curve often finishes at a high level although the resting juice has a low acidity but it must be remembered that the latter is taken in the early morning twelve hours or more after a meal. In addition, the secretion of appetite juice in expectation of a meal at the end of the test probably plays a part in the formation of the terminal portion of the curve.

The variations in this normal picture which are induced by ulceration in the stomach and duodenum are as follows —

A Gastric Ulcer—Under this heading are included chronic ulcers occupying the body of the stomach. Three cases of this type have been examined, and show certain features in common. An example of the type of curve obtained by the fractional test meal in this condition is shown in *Chart IV* (Fig. 321).

1 Motility—All test meals showed marked delay in the passage of food from the stomach, confirming the x-ray reports on these cases, which stated, 'marked delay at the pylorus, probably due to reflex spasm'. In one case, in which the stomach was greatly dilated and atonic, starch was present in the resting juice and remained throughout the test (three and three-quarter hours).

2 Bile—Bile was present over much longer periods than is usual in the normal stomach, and in the case referred to above was found in every specimen.

3 Secretion—Whilst no characteristic shape can be ascribed to the curve, its essential feature is subacidity, ulcers of the body being associated with hyposecretion.

As far as the secretory function is concerned, one would hesitate to make a diagnosis of gastric ulcer on the evidence of the fractional test meal alone. Although blood was not found in these specimens, its appearance in cases of active ulcer is to be expected, and would afford additional evidence for diagnosis. If the clinical history as regards pain etc., leads one to suspect a gastric ulcer, a fractional test

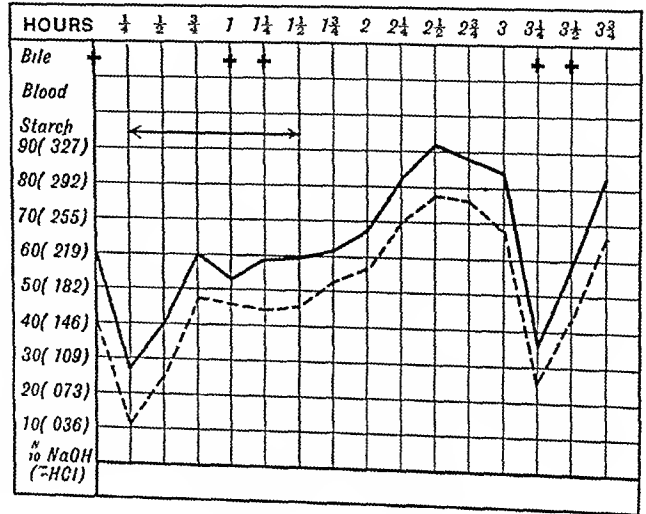


FIG. 322—Chart 1 Duodenal ulcer

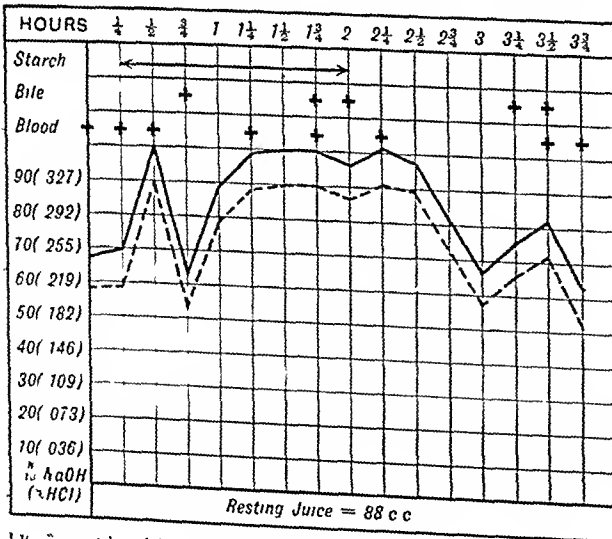


FIG. 323—Chart 11 Duodenal ulcer with very high acidity and haemorrhage

meal showing subacidity associated with delayed emptying is distinctly confirmatory.

B Duodenal Ulcer—The alterations in the gastric functions associated with chronic duodenal ulcers and is displayed in a fractional test-meal chart are very typical (Charts I and II (Figs. 322, 323) show the curve in this condition).

1 *Motility*—The earlier disappearance of starch from the gastric contents affords evidence of 'hurry' in the majority of cases. This has been noted even when a ray examination has reported delay. Oatmeal provides a more natural food than the bismuth mixture necessary for a ray examination, and therefore the fractional test meal gives us a more accurate picture of the gastric motility.

Examination of the ulcer at operation sometimes proved that either the ulcer itself or the surrounding fibrosis encroached upon the pyloric ring, and in these patients the rate of emptying was less rapid, approximating to, but never more delayed than, the normal.

2 *Bile*—Occasionally present in the resting juice bile enters at frequent intervals during the meal.

3 *Secretion*—The resting juice never less than 40 c.c. in amount, is highly acid. The average level in this series was 50, with a free acid reading 10 to 15 below. The acid curve is typically high, displaying as a rule a number of peaks. The curves of free acid and total acidity run parallel. The maintenance of a high level of acidity after the stomach has emptied itself of food should be noted.

It is usually at this time that the highest degree of acidity is reached. This fact, combined with the high acidity of the resting juice, indicates a condition of continuous gastric hypersecretion associated with duodenal ulcer.

C Pyloric Ulcer—In this class are placed all ulcers occurring in the pyloric portion of the stomach, including the pyloric canal and vestibule, as it has been found that the associated disturbance of gastric function gives rise to a fractional test-meal curve which enables these ulcers to be differentiated from those situated in the body of the stomach or in the duodenum. A typical curve is represented by Chart VII (Fig 324).

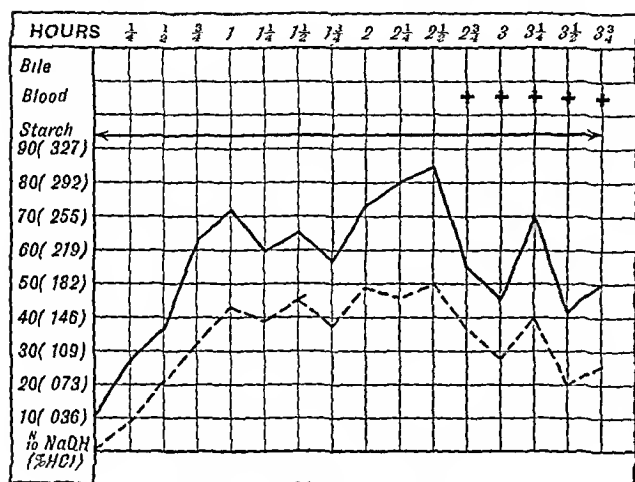


FIG. 324.—Chart VII. Pyloric ulcer.

1 *Motility*—Delay in emptying is invariably found. This delay is often attributed in a ray examination to obstruction at the pylorus, when operation shows that the pylorus is free. In these cases the cause is possibly a reflex spasm of the canal or pars pylorica similar to the hour-glass spasm seen in ulcers of the body of the stomach. This contraction is seen in the ray examination and taken for the pylorus, which is actually some distance away on the duodenal side. Reflex spasm of the pylorus itself may also account for the delay.

2 *Bile*—A feature of ulcers in this situation is the comparative absence of bile from the stomach. When present it appears late in the meal.

3 *Secretion*—The resting juice is of smaller quantity than is found in duodenal ulcer (unless a marked degree of pyloric stenosis be present), and, unlike the latter shows an acidity well within the normal limits. From this low level the curve rises, and shows a considerable degree of hyperacidity, which is maintained for a time, but tends to fall before the end of the test. In some cases, as in the example shown, the HCl curve does not show the same tendency to run parallel with the curve of total acidity that is seen in other conditions. The retention of the products of gastric digestion allows a greater combination of protein and HCl to take place, thus relatively increasing the amount of total acidity. Two of the curves obtained were of the 'climbing type'.

described by Bolton⁹ as due to spasm of the pylorus. In view of the smaller quantity found of resting juice of low acidity, it must be assumed that hyper-secretion is not so marked a feature with pyloric as with duodenal ulcers, and the excessive secretion occurs as a reaction to a food stimulus rather than as a continued independent phenomenon.

Included in the group under consideration are ulcers situated actually at the pylorus. It is a matter of the greatest difficulty to declare definitely, at the time of operation, the exact source of these ulcers. One is faced with a scarred ulcer mass whose cicatrix and surrounding oedema have obliterated to a large extent the usual landmarks—the pyloric vein and the faint pale line that mark the pylorus in the normal stomach. There are only two possible sources of origin. Either the ulcer has occupied the duodenopyloric fornix¹¹ and grown into the sphincter, or it has begun in the termination of the pyloric canal itself. Whatever their origin, the fractional test meal shows that their secretory curve approximates more closely to the duodenal type in displaying a larger quantity of resting juice of high acidity, but the associated motor signs are those of ulcers on the gastric side of the pylorus. The delay in these cases is due to cicatrization and oedema rather than to spasm.

Though certain degrees of hyperacidity may be found in conditions other than ulceration of the stomach and duodenum the typical features of the curves described above have not been met with in such conditions. In three patients who gave curves of the type described, the scar of a healed ulcer was found producing no obstruction or deformity. It thus appears that the abnormal functional state of the stomach can persist after the healing of the ulcer.

With regard to the question as to whether hyperacidity is a cause of pain in ulceration, a study of the curves showed no constant relation between the times at which the gastric acidity reaches its highest levels and the time of onset of the pain unless one can associate the onset of late pain in duodenal ulcer with the entrance into the duodenum of the highly acid after secretion.

EFFECTS OF GASTRO-ENTEROSTOMY IN THE DIFFERENT TYPES OF ULCER

After this preamble we may discuss in more detail the effects of gastro-enterostomy on the function of the stomach in the above-enumerated pathological conditions.

1 Motor Effects—As already mentioned, the rate of emptying after a satisfactory gastro-enterostomy is always considerably more rapid than is found in the normal stomach. The average time taken by the stomach to empty itself of food under the new anatomical conditions is one and a quarter to one and a half hours, and this is irrespective of the position of the initial ulcer. With ulcers at or on the gastric side of the pylorus this means that the food leaves the stomach at a very much earlier time than was possible with the pre-operative delay. When the pylorus is free one would expect to find that the additional opening increased the speed of emptying but this view does not take into account the effect of the chemistry of the stomach and duodenum in exercising a control over the movements of these organs. Thus, in duodenal ulcers associated with marked hurry before operation, a gastro-enterostomy lowers acidity and sometimes diminishes the rate of emptying although this remains more rapid than in the normal unoperated stomach. Although the fractional test meal supplies no direct evidence on the question the size of the stomach is probably not a matter of very vital importance, for we may assume that nowadays all surgeons make the gastro-enterostomy opening at least larger than the normal pyloric diameter.

Five test meals were carried out on patients in whom partial gastrectomy had been performed. They showed a still faster emptying than is seen after simple gastro-enterostomy. The average time was three-quarters of an hour. It is noteworthy that in several patients examined whose symptoms of pain and vomiting had recurred after gastro-enterostomy the stomach took two or more hours to empty. In one of these, at least the stomach had ceased to function as an exit for food.

2 Effects on Gastric Acidity—Assuming that similar anatomical conditions are present in all cases after gastro-enterostomy, the extent to which the gastric acidity is reduced must depend on the relative quantities of acid in the stomach and of alkali entering through the stoma

We have no reason to suppose that the latter varies with the position of the ulcer, but we have already seen that the quantity of acid secreted by the stomach does vary according as the ulcer is nearer the cardia or the duodenum

The fractional test meal confirms what might be expected from the theoretical consideration of the effect of gastro-enterostomy in ulcers in these different situations. It may be stated at the outset that whether partial gastrectomy or gastro-enterostomy alone be performed there appears to be no difference in effect on the resulting acidity. In the cases in which the former operation had been performed

the post operative reduction in acidity was no different from what one would expect from gastro-enterostomy alone

a Gastric Ulcer—The state of hyposecretion and hypo acidity associated with ulcers of the body of the stomach favours a large reduction in the post operative acidity. In all cases free acid entirely disappeared from the stomach, and the total acidity did not rise above 12. *Chart VIII* (Fig 325) shows the post-operative curve of the case whose curve before operation is seen in *Chart IV* (Fig 321)

b Duodenal Ulcer—Since duodenal ulcers are accompanied by such marked hypersecretion, a much smaller reduction in acidity after gastro-enterostomy is to be expected. It has been found invariably that a considerable degree of acidity exists after operation, and a study of the acid curves shows that a reduction to about half of the pre-operative acidity is the maximum that has been obtained. That is, the gastric secretion is now brought within the limits of normal acidity. An example of this is shown in *Chart IX* (Fig 326). In a minority of cases a condition of hyperacidity persists after operation as can be seen in *Chart X* (Fig 327), although the high acid level is not now maintained over such long periods, and the acidity of the resting juice is always low. These curves

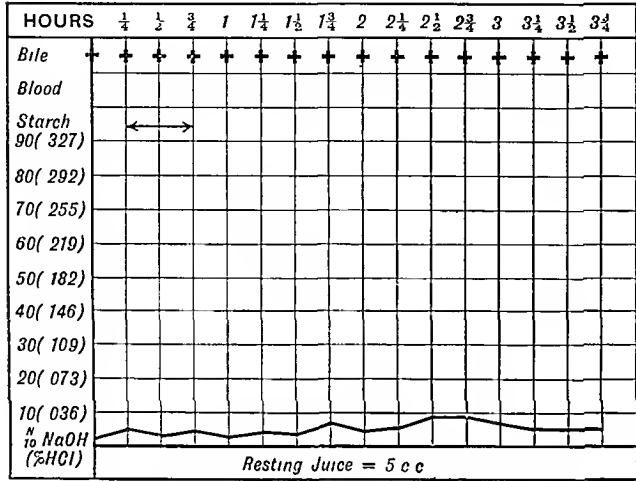


FIG 325—Chart VIII Gastric ulcer after gastro-enterostomy. Pre operation curve shown in Chart IV (Fig 321)

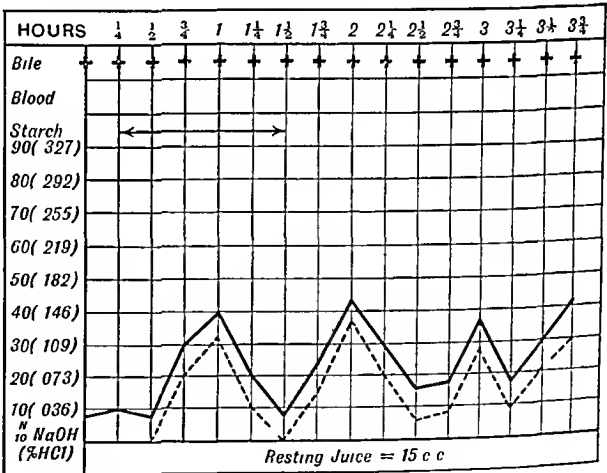


FIG 326—Chart IX Duodenal ulcer after gastro-enterostomy with maximum reduction of acidity. Same case as in Chart VIII (Fig 325)

show a large number of sudden drops in acidity reaching nearly to the base line. This probably indicates the influx of quantities of pancreatic juice at frequent intervals.

In connection with the small reduction of acidity effected by operation it is interesting to note the much greater frequency with which jejunal ulceration follows gastro-enterostomy performed for duodenal ulcer than when performed for ulceration definitely on the gastric side.

c. Pyloric Ulcer—The gastric hypersecretion associated with these ulcers is less than that of duodenal ulcer, and a correspondingly greater reduction of acidity is shown after operation. Chart XI (Fig. 328) shows a typical example. The reduction is to a subacid level, but free HCl always appears at some period of the meal.

It will be seen from the above observations that the evidence of the fractional test meal is distinctly in favour of the drainage theory as an explanation of the beneficial effects of gastro-enterostomy in chronic ulceration of the stomach and duodenum, since a clinically satisfactory result may be obtained even in the presence of a high degree of post-operative acidity.

The association of delayed emptying after operation with a recurrence of symptoms of pain and vomiting has already been mentioned.

Of the cases examined in which gastro-enterostomy had provided but little relief of symptoms, the majority showed an absence of free acid and a very low total acidity. Two of these were operated on again, and no trace of an ulcer was found, one being the case of achlorhydria referred to above. Two cases of this condition and one of achylia gastrica were met with. They presented symptoms very suggestive of chronic ulceration, and in one of them x-ray examination in two different hospitals had led to a diagnosis of ulcer being made—a diagnosis that could have been excluded by

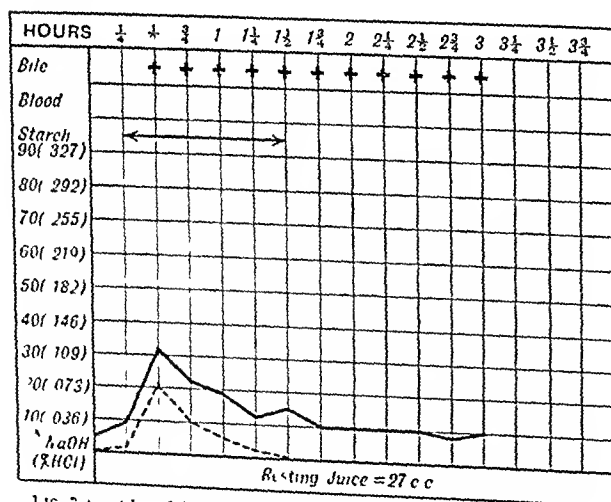


FIG. 328.—Chart XI. Pyloric ulcer after gastro-enterostomy. Same case as in Chart III (Fig. 324).

a preliminary examination with the fractional test meal.

The view expressed by Sherrin¹⁰ that duodenal exclusion is the cause of post-operative hyperacidity is not borne out, as it has been demonstrated that a high acid level after operation is but the natural result of an excessive pre-operative hypersecretion and is not affected by variations in operative procedure apart from the provision of a stoma.

SUMMARY

- 1 In performing a fractional test meal the withdrawal of small quantities of gastric juice and their immediate filtration are essential to an accurate result
- 2 Ulcers of the body of the stomach are associated with delay and hypo acidity, duodenal ulcers with hurry and hyperacidity
- 3 Ulcers of the pyloric portion of the stomach show a disturbance of gastric function intermediate between these
- 4 Bile is invariably found in the stomach after gastro enterostomy
- 5 After a satisfactory gastro enterostomy, food always leaves the stomach more rapidly than is normal
- 6 The post-operative reduction in acidity is greatest in gastric ulcer and least in duodenal ulcer, in which a condition of hyperacidity may remain
- 7 The evidence of the fractional test meal supports the drainage theory of gastro enterostomy

In conclusion, I wish to express my gratitude to Mr Geoffrey Jefferson, who has been responsible for most of the operative work involved in this investigation, for his assistance in obtaining the results quoted, and for the many useful suggestions he has offered

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CONGENITAL CYST OF THE COMMON BILE-DUCT: WITH REPORT OF TWO CASES

By JOHN WORLEY, MANCHESTER

THE following are the notes of the two cases on which this paper is based —

Case 1—E R, a girl, age 17, by occupation a domestic servant, was admitted to St Mary's Hospital, Manchester, on November 1, 1921, under the care of Professor W E Fothergill, suffering from abdominal pain and swelling.

HISTORY OF PRESENT ILLNESS—Two years before admission she began to suffer from attacks of pain in the right abdomen. The pain was described as sharp in character, as though something was running into her side, and during the past two years she had rarely passed a day without feeling it. The pain was usually at its worst about half an hour after food, and about one hour after a meal she would often vomit with temporary relief from pain. The vomit was never bile stained. For several weeks before admission she had noticed that her abdomen was growing larger. Six weeks before admission, after an unusually bad attack of pain and vomiting, the whites of her eyes were yellow for two or three days, but apart from this she had never been jaundiced. She had never noticed the colour of her motions. She had been constipated since her illness began. There had been no loss of weight. Her family doctor sent her to St Mary's Hospital as a case of (?) ovarian cyst.

PREVIOUS HEALTH—She had convulsions when three weeks old, and has had slight athetoid movements of the hands ever since. Her mental development is slightly defective. Otherwise she has always had good health until the onset of the present trouble.

FAMILY HISTORY—She was the fourth of a family of fifteen. The three children born before her (two male and one female) all died jaundiced, at the age of one month, one week, and two days respectively. One other child, born later, has died at the age of nine months, but without jaundice. The cause of these deaths was not ascertained by post mortem examination. The rest of the family are healthy.

CONTRIBUTION ON ADMISSION—The girl's nutrition was moderately good, and she was not jaundiced. Considerable abdominal distention was obvious on inspection, and palpation showed a round smooth swelling, tense and cystic which almost filled the abdominal cavity, with its centre to the right of the mid-line (Fig 329). The tumour was dull on percussion. There was resonance in the left flank and left iliac fossa. On pelvic examination the uterus was normal and appeared to have no connection with the cyst. The faeces were clay coloured, and the bowels considerably constipated.

INTRA-OPERATION—Professor W E Fothergill explored the abdomen through a vertical incision in the right rectus. A huge cyst was found extending down from under the liver. It was sutured to the peritoneal parietum at the edges of the wound and inserted into the wound and incised, when many pints of thin green bile escaped. A drainage tube was inserted into the cyst. The tube was removed after a few days. A drainage tube was inserted into the cyst. The tube was removed after a few days. Bile continued to escape through the fistula in large quantities, but at the end of three weeks the fistula had nearly closed, and it was evident that the cyst was filling again. During the whole of this period the faeces were clay coloured.

SECOND OPERATION Nov 22—[The case is given by Dr A W Mitchell. The abdomen was opened by a vertical incision in the right rectus. The previous one and surrounding the biliary fistula at its lower end. On opening the peritoneum above the fistula, a small empty gall bladder with pointed fundus was seen projecting below the margin of the liver. Below the liver and adherent to the interior abdominal wall at the site of the fistula was a large round cyst with thick opaque walls which now contained some two pints of fluid. The cyst was freed from the abdominal wall and the fistula clamped. During this procedure several ounces of pure bile escaped. Examination of the relations of the cyst demonstrated that it was in enormous saccular dilatation of the common bile duct. The pylorus and first part of the duodenum were

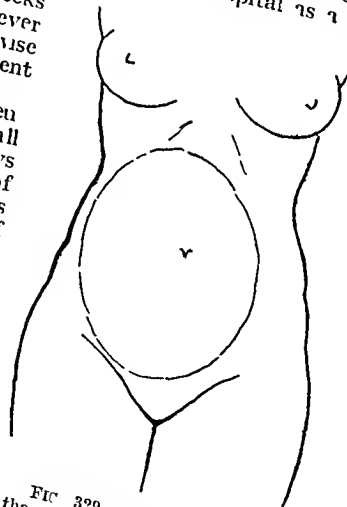


FIG 329—Diagram to show the extent of the tumour before the first operation

stretched across the anterior and lower aspect of the cyst, and bound down to it by peritoneum

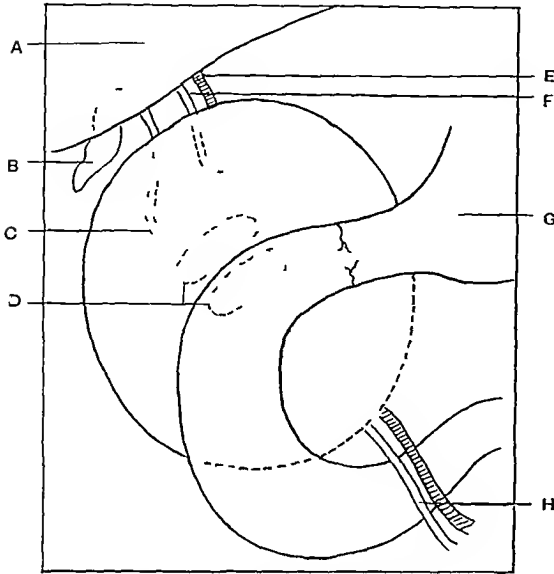


FIG. 330.—Diagram showing relation of parts at second operation. (A) Liver (B) Gall bladder (C) Cystic duct (D) Site of anastomosis (E) Hepatic artery (F) Hepatic duct (G) Stomach (H) The superior mesenteric vessels

(Fig. 330) The second part of the duodenum turned downwards in the normal manner, and only its upper two thirds lay in intimate relation with the cyst. The duodenum was decidedly distended from the pylorus as far as the point where the superior mesenteric vessels crossed its third part. Beyond this it was contracted. The downward thrust of the cyst on the root of the mesentery appeared to have caused a partial obstruction of the third part of the duodenum between the superior mesenteric vessels and the spine, after the manner of a chronic duodenal ileus.¹

A stomach tube was passed at this stage in order to get rid of the trouble some distention of the stomach and upper duodenum. The hepatic flexure of the colon was displaced downwards by the cyst and lay completely below it. As there was evidently no adequate outflow from the cystic dilatation of the common duct into the duodenum, and as the first part of the duodenum lay in easy apposition with the cyst, it was decided to perform a choledochoduodenostomy. This was done by two rows of fine catgut with the aid of clamps. The cyst wall was quite as thick as that of the duodenum, but tough and fibrous, so that the needle was drawn through it with some difficulty. The clamped external fistula was next packed round with swabs and the clamp removed,

when rather more than a pint of green watery bile escaped. The fistulous tract was excised, the opening into the cyst enlarged, and its cavity swabbed dry. It appeared to be lined by a pale mucosa which was intimately adherent to the tough fibrous wall. The slit-like orifice of the cystic duct could now be seen in the right and upper region of the cyst, and a small probe inserted into it passed readily into the gall bladder. The opening of the hepatic duct into the cyst was not definitely made out, but it was situated more posteriorly and to the left as far as could be made out by external palpation. The hepatic and cystic ducts were not dilated.

The opening from the cyst via the lower part of the common bile duct into the duodenum could not be found though—judging by the absence of icterus—it must have been more or less patent. The stomach from the cyst into the duodenum was examined, and was large enough to admit a finger readily. A small piece of the wall of the cyst was excised for microscopic examination, and the opening in it was sutured in two layers. The abdominal wall was closed without drainage.

SUBSEQUENT PROGRESS.—Bilious vomiting was very persistent for the first week after operation, and caused so much anxiety that I considered the advisability of dividing the stomach at the pylorus, closing the distal end, and implanting the proximal end into the side of the first jejunal loop as in a Polya gastrectomy. From the seventh day, however, the vomiting ceased, and the patient's condition

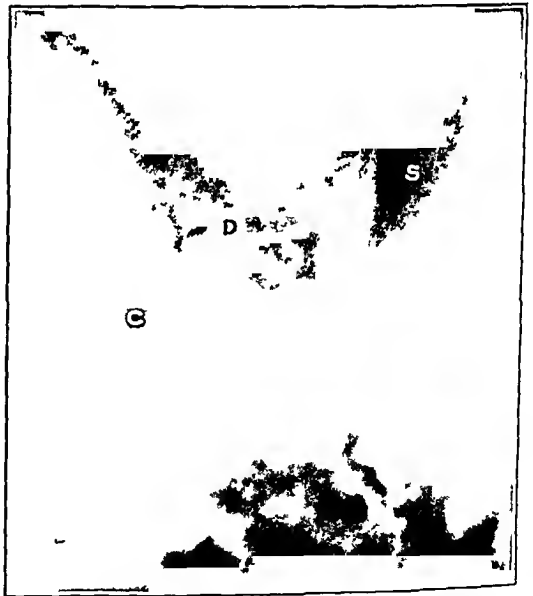


Fig. 331.—Radio-gram showing cavity of cyst containing barium after choledochoduodenostomy. (D) Duodenum (C) Liver in cyst (S) Barium pushed into upper part of stomach

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improved rapidly. The wound healed by first intention. Three weeks after operation an x-ray examination after a barium meal was made in order to ascertain whether there was any regurgitation of duodenal contents into the cyst through the stomach. The result is shown in Fig 331, for which I am indebted to Dr Barelay. Barium is shown passing from the first part of the duodenum through the stomach into the cyst, which is definitely outlined. It has diminished in size considerably. The patient's subsequent convalescence was uneventful, and she reported in November, 1922, that she had been in good health since her discharge from the hospital.

I am indebted to Dr H T Ashby and Mr H Platt for notes of the following case, which was under their care in the Manchester Children's Hospital.

Case 2—H M, a girl, age 6½ years, was admitted to hospital on September 23, 1915, on account of jaundice, abdominal pain, and wasting. **HISTORY**—The child had always been delicate, thin, her skin and sclerotics were yellow, while the stools were pale in colour. The child had complained of intermittent abdominal pain during the same period and had lost weight considerably. **FAMILY HISTORY**—The child was one of a family of seven, of whom two children had died in infancy of malnutrition.

CONDIION ON ADMISSION—The child was moderately well nourished and deeply jaundiced. In the upper right abdomen was a large, smooth, rounded swelling, not tender on palpation, extending from the costal margin down to the umbilicus, and apparently continuous with the liver. There were no signs of fluid in the peritoneal cavity. The swelling was considered to be a cyst, originating in the liver. X-ray examination showed no abnormality in the chest, and both domes of the diaphragm were at the usual level, and moved normally on respiration. The blood examination showed 4,500,000 red cells per cmm and 8500 leucocytes. Two days after admission the jaundice was noticed to be disappearing, and in a few more days had gone entirely. The tumour diminished in size at the same time, and the child's general health improved. **FIRST OPERATION**—An exploratory operation was undertaken by Mr Platt on Oct 23. A vertical incision was made through the upper part of the right rectus. The liver and gall bladder were found to be normal, and immediately below them a large cyst was found in the position of the common bile duct. A trocar was inserted and a large quantity of bile escaped. A drainage tube was then inserted into the cyst, and the wound closed up to it. The child's progress for the first few days was satisfactory, but ten days after the operation she showed signs of exhaustion resulting from the profuse discharge of bile from the wound. She improved to some extent after this, though the biliary fistula persisted, and the feces remained clay coloured. **SECOND OPERATION**—On Dec 22, two months after the original operation, a second operation was undertaken for the purpose of closing the biliary fistula. The child sank and died a few hours later. No post mortem examination could be obtained.

Frequency of Cyst of the Common Bile-duct—Idiopathic cyst of the common bile-duct is a condition of considerable rarity. Erik Waller,² who published an exhaustive account of the condition in 1917, was only able to collect 35 cases, including one of his own. Since the publication of Waller's paper, I have only been able to find other cases recorded by Kremer,³ Budde,⁴ McConnell,⁵ and Reel and Burrell,⁶ making a total, with my own cases, of 41 recorded cases.

Pathological Anatomy—A study of the recorded cases proves that the disease presents anatomical features that show very few variations of any importance. There is a cystic dilatation of the upper end of the common bile-duct which enlarges slowly and progressively, much like a saccular aneurysm. The hepatic and cystic ducts may open separately into the cyst, as in my own case. The intrapancreatic and the terminal or intramural portions of the common duct are not involved in the cystic dilatation, and in all cases in which the point has been investigated the lower end of the common duct below the cyst has been found to be patent. In Budde's case⁴ Vater's ampulla was placed on the front and right aspect of the duodenum. That obstruction to the flow of bile does occur is evident from the frequency with which jaundice is recorded. The obstruction is however often intermittent. Periodical diminution in the size of the cyst has been observed in some cases, with consequent remission of the jaundice. The patent lower end of the common duct usually lies in the medial wall of the cyst, and it would appear that the distention of the cyst gives rise to a saccular obstruction to the common duct at the point where it is suddenly reduced to its normal calibre. The saccular nature of the dilatation, with normal ducts above and below the site forms a striking contrast with the diffuse dilatation of the bile-duct met with in a secondary result of gall stone obstruction or compression of the common duct by pancreatic tumours.

The size of the cyst varies in different cases, but it is commonly described as the size of a cocoa-nut or a man's head. In my own case (*Case 1*) the cyst so nearly filled the abdomen that the first doctor the patient consulted made the diagnosis of advanced pregnancy. As the cyst enlarges, the first part of the duodenum is sometimes pushed downwards, while the anterior layer of the gastroleptic omentum is stretched over the anterior wall of the cyst. In other cases, as in my own, the pylorus and duodenum are stretched across the anterior wall of the cyst, to which they are bound down by peritoneum (*Fig 330*). The hepatic flexure is usually thrust downwards and to the left by the enlarging cyst.

A feature in my case, which has not been recorded in others, was the marked obstruction of the third part of the duodenum by compression behind the superior mesenteric vessels, due to the downward thrust of the cyst on the small intestine and root of the mesentery. It is possible that this condition of secondary duodenal ileus was responsible for the frequent gastric pain recurring an hour or so after food.

The gall-bladder has generally been found to be more or less empty, as in my case, though sometimes it contains enough bile to form a small palpable swelling immediately above the large cyst. In some advanced cases the liver has been involved in biliary cirrhosis, secondary to the obstruction of the duct. The portal vein lies behind the cyst, and the hepatic artery is displaced to the left. The tendency of the cyst as it expands is to grow, from the site of its origin in the supraduodenal or retroduodenal portion of the common duct, downwards and to the right, so that the normal lower end of the duct comes to lie in the left or medial wall of the cyst. This direction of the growth is probably the line of least resistance under the mechanical pressure of the surrounding viscera, notably the liver, which prevents any marked expansion upwards. It will be noted that the cyst primarily encroaches on the subhepatic fossa of the peritoneum, or right kidney pouch.

Structure of the Cyst Wall—The thickness of the cyst wall varies considerably in different cases. In some recorded cases it has ruptured at once on manipulation by the surgeon, in others it is described as thick, tough, and opaque. In my case it was about as thick as the duodenal wall, but much tougher, so that it was a matter of some difficulty to push an intestinal needle through it. It was yellow in colour and opaque. Transverse sections of the cyst wall after fixing and staining were 2.5 mm thick. The histological structure as seen in transverse section was as follows. The mucous membrane lining the cyst had entirely disappeared from the portion examined and the wall was composed of dense fibrous tissue, with a layer of endothelium on its outer aspect where the peritoneum was adherent to it. A similar absence of the lining mucous membrane was reported by Kremer³ in sections from his case. In the case reported by Reel and Burrell⁶ the cyst was lined by a single layer of columnar epithelium, and the wall contained some isolated lobules of liver cells. Since the cyst was adherent to the liver in this case, it is possible that the lobules may have been stripped off from the liver during separation of the cyst.

Etiological Factors —

Age—The average age at which symptoms first arose was from 12 to 14 years in Waller's series of 35 cases. A typical cyst of the common duct, 3 by 2.5 cm in size, was noted by Heilger⁷ in a full-term foetus. It was associated with a congenital diaphragmatic hernia. Olex⁸ in 1883, also reported a case in an infant, age five weeks, in whom the swelling had been noticed from birth. These two cases afford convincing evidence that the condition originates in some congenital defect of the duct. That the enlargement of the cyst is sometimes only very slowly progressive is proved by the case of Reel and Burrell⁶ whose patient, age 56 when operated on, had noticed the swelling from the age of 20. It would appear that the cyst may enlarge slowly for several years without producing symptoms and that the characteristic symptoms pain and jaundice, are only produced when the cyst has reached such a size as to cause mechanical obstruction to the duodenum and lower part of the common duct.

Sex—Of the 41 recorded cases, 36 occurred in females, a percentage of 88 females to 12 males. This marked preponderance of females shows in interesting parallel with the sex relationship in congenital dislocation of the hip and its explanation is equally obscure.

Hereditarily—There is little evidence that hereditary influences bear any part in the etiology of the condition. It is suggestive that the three children in the same family who

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were born before my patient all died, jaundiced, at the age of one month, seven days, and two days respectively, but in the absence of post-mortem examinations this evidence can hardly be regarded as of any scientific value.

We have, therefore, to deal with a localized dilatation of the common bile-duct, congenital in origin, and very slowly progressive. Its precise mode of origin and its causation remain obscure. A study of the embryological accounts of the formation of the liver and bile-ducts as an outgrowth from the hypoblast of the intestinal canal at the junction of the foregut and midgut does not throw any light on the formation of this rare anomaly.

In Budde's case,⁴ the opening of the common bile-duct into the duodenum was not at the usual position on the postero-internal aspect of the second portion, but more on the right and anterior aspect of this part of the duodenum. This position of Vater's ampulla would appear to indicate some abnormality in the rotation of the midgut loop and peritoneal fixation of the duodenum, which may prove to be the prime cause of the dilatation of the bile-duct. The point requires further attention, and wherever possible in future cases the precise position of the lower end of the common duct should be recorded. Budde considers that these cysts are due to congenital dilatation of the extraduodenal part of the common duct, increased by valvular obstruction at the lower end, and by inspissation of bile. He ventures the suggestion that the primary diverticulum is due to a pancreatic dilatation of the duct. This dilatation increases progressively as a result of the tension of its contents. This conception is based on the analogy of the well-known diverticulum of the duodenum, which often contain pancreatic tissue. There does not appear, however, to be any solid histological foundation for his ingenious theory, since in no recorded case of choledochus cyst has pancreatic tissue been found.

Symptoms and Signs—The clinical manifestations consist of attacks of abdominal pain, associated with a tumour, and usually with jaundice. The attacks of pain recur at varying intervals, but tend to grow more severe and more frequent as the cyst enlarges. The pain varies in intensity in different cases. In some it is described as a feeling of flatulence, or acute indigestion, is worse half an hour or so after meals, and is usually relieved by vomiting. In others there are less frequent but more severe attacks of a colicky or spasmodic nature. The pain is usually referred to the epigastric region. It has been suggested above that the pain may be due, in some cases at least, to partial obstruction of the third part of the duodenum by the superior mesenteric vessels.

The tumour varies greatly in size. When small it lies under the upper right rectus. When very large it may fill almost the entire abdomen except the right iliac fossa. Fluctuations in size have been recorded, and the cyst has been noticed to be more tense and prominent after meals in some cases. In others there have been periods in which the tumour has become smaller, and the pain and jaundice less severe, or absent, but in spite of these occasional remissions, the tumour tends gradually to enlarge.

Owing to the position of the cyst immediately below the liver, the liver margin is thrust up under the lower ribs and can seldom be palpated, though cases are recorded in which a distended gall-bladder has formed a palpable swelling immediately above the cyst. The dullness of the tumour on percussion is continuous with that of the liver, but on account of its peritoneal fixation the cyst does not move up and down so distinctly on respiration as does in intrahepatic cyst.

Jaundice has been a marked feature in the great majority of recorded cases. In Waller's case and in my own there was only slight tingling of the sclerotics for a short time, but much more commonly it has been quite unmistakable for some time before operation. In one of the cases the condition is undoubtedly responsible for the lack of portal vein or adenoma from pressure on the inferior vena cava, has occurred.

Diagnosis—The great rarity of the condition is undoubtedly responsible for the lack of a correct pre-operative diagnosis in any recorded case. The association of a typical subhepatic cystic swelling with jaundice and attacks of epigastric pain in a girl of 14 to 20 years should lead one who is familiar with the literature to a correct diagnosis. The physical signs are not easy to distinguish from those of hydatid cyst of the liver or pancreatic cyst. The former, however, rarely produces pain and moves more freely.

on respiration and on palpation than does a cyst of the common bile-duct. Examination of the liver by x rays after inducing pneumoperitoneum, would probably demonstrate the intrahepatic situation of the hydatid cyst. Pancreatic cysts, with the exception of pseudocysts in the lesser sac of the peritoneum, seldom attain a size comparable with that usual in cyst of the common bile duct, and the pseudocysts are invariably preceded by severe trauma. It must be admitted, however, that a cyst arising in the head of the pancreas might produce symptoms and physical signs that could not be distinguished with any certainty from cyst of the common bile duct.

Treatment and Prognosis—Two factors have combined to give a deplorably and quite unnecessarily high death-rate to the cases that have hitherto been operated upon. In the first place, operation has been deferred until the patient has become profoundly ill from jaundice, with pain and vomiting, and is little able to stand a major operative procedure. In the second place, and more important, the operating surgeon has too often been unfamiliar with the condition, and has consequently adopted inappropriate measures. Three cases have been treated by extirpation of the cyst through failure to recognize that the 'cyst' was an essential though abnormal part of the common bile duct. These cases were inevitably fatal. Twenty-two cases have been treated by incision and external drainage. Of these, 20 have died (one survived three years with a fistula, and died of phthisis) while 2, Reel and Burrell's case⁶ and McConnell's case⁵, were in good health when the cyst was reported, though one required secondary drainage of an abscess in the sac. This abscess apparently led to shrinking of the cyst. The formation of an external biliary fistula plainly affords very little hope of relief for the patient. It is less disastrous than extirpation of the sac, but a permanent leaking of bile from the wound is a deplorable prospect for the patient. Moreover, the fistula tends to close, when the swelling of the cyst will usually recur.

The only rational treatment consists in the formation of a permanent anastomosis between the dilated bile duct and the alimentary canal. The stomach, duodenum, or jejunum may be utilized, but a fistula with the duodenum gives the nearest approximation to the normal, and is to be preferred, as it presents no great technical difficulties. Of 3 cases in which a primary anastomosis was made, 2 recovered and 1 died. In the fatal case the cyst was drained externally at the same time. Primary drainage with choledochenterostomy as a secondary operation, was performed in 8 cases. Of these, 5 recovered and 3 died, but in the three fatal cases it would appear that the attempt at anastomosis was not altogether successful.

Primary lateral choledochenterostomy, without drainage, would appear to be the operation of choice. It is necessary to evacuate some of the contents of the cyst in order to effect the anastomosis, but this can readily be done with an ovarian trocar or a large exploring syringe. Temporary external drainage of the cyst may be adopted as a palliative measure if the patient is in a critical condition, but the prospect of lasting relief from drainage is remote, and the anastomosis should be undertaken as soon as the patient's condition warrants it. In the event of severe post operative vomiting, division of the pylorus, with invagination of its distal end, and implantation of the proximal end into the side of the first jejunal loop, may be considered, but it is certainly not required as a rule.

I am indebted to my colleague, Professor W. E. Fothergill, both for the opportunity of operating on the case herein recorded, and for the two diagrams illustrating the condition.

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SHORT NOTES OF
RARE OR OBSCURE CASES

ACUTE HÆMORRHAGIC PANCREATITIS
PANCREATIC DUCT
ROUND WORM IN

By Sir HUGH V RIGBY, K.C.V.O., London

The patient, Annie Barker, a domestic servant, single, age 30, was admitted to the London Hospital on Jan 21, 1909 with the following history

On the previous day, whilst at work, she was seized with sudden severe pains in the abdomen. The pain was cutting in character and was chiefly located across the lower part of the abdomen and in the back. She vomited ten minutes after the onset of pain, and, during the rest of that day, the pain continued and she had constant vomiting and shivering attacks.

To day (Jan 21) she still complains of pain, chiefly in the left iliac fossa. The sickness has stopped, though she still has nausea. The bowels have not acted for two days. In the evening, her general condition was grave.

PHYSICAL SIGNS—Patient not very fat, there is no note of any marked cyanosis. Pulse 120, small and rather irregular. Respiration 30. Temperature 100°. Tongue dry. Inquiries elicit no history of previous gastric trouble. Menstrual history normal.

On examining the abdomen, no appreciable distention can be made out. The abdominal wall is rather rigid, there is some movement of the lower abdomen on respiration, superficial tenderness and pain on palpation, most complained of over the lower abdomen. There is no evidence of free fluid in the peritoneum.

DIAGNOSIS—Not clear, but seemed to lie between acute appendicitis and perforated gastric or duodenal ulcer. Operation was carried out soon after admission.

OPERATION—Abdomen was opened by a median incision below the umbilicus. A quantity of reddish, odourless fluid escaped, and a hurried examination of the appendix region and pelvic organs revealed no evidence of disease. A tube was passed down to the pelvic floor and the incision covered up.

A second incision was then made above the umbilicus in the middle line. A quantity of similar reddish fluid escaped, and then extensive fat necroses were seen scattered about on the omentum and mesentery. The lesser sac was now opened up by tearing a way through the lesser omentum, still more reddish fluid escaped in this region. The pancreas was then seen to be greatly swollen, it was mucous coloured and mottled on the surface, and the substance was soft and sponge like. Its interior surface was scratched through.

The cavity of the lesser sac was washed out with boric acid, and gauze tampons were pressed down to the surface of the pancreas. The gall-bladder and region of the common duct were examined. There was no obvious sign of disease, so the gall-bladder was not drained. Both abdominal wounds were partially closed, a drainage tube being left pressed down to the floor of the pelvis.

The patient stood the operation fairly well. Rectal saline was given, and during the night a large quantity of fluid drained away from both wounds. Next morning her condition was not improved. She still had some pain—not very severe. Temperature 101°. Pulse 120. Towards evening her condition became much worse. She had incontinence of urine. Pulse weaker 140 per minute. Respirations 44 to 50 per minute. She died suddenly at 10 p.m. after vomiting.

Before the operation, a specimen of urine had been saved for examination. Analysis showed a trace of albumin, but no sugar. Cammidge's pancreatic test was positive.

The fluid removed from the abdomen was examined, and found to be faintly alkaline and to contain an active starch-splitting ferment—very slight digestive power on albumin. Deposit of the fluid contained blood and numerous pus cells—no organisms in films. Cultures sterile.

POST-MORTEM EXAMINATION—The following is an abstract of the notes—

Macroscopic diagnosis. Haemorrhagic pancreatitis. Fat necroses were found throughout the peritoneum, including the under surface of the diaphragm. Operation drainage tubes in lesser sac and in the pelvis. Cloudy, sanious fluid in the lesser sac.



FIG. 332.—Round worm found post mortem in pancreatic duct. (D) Duodenum. (A) Worm in duct. (S) Stomach turned up. (H) Hemorrhage in pancreas.

Some pus in the pouch of Douglas. Hyperemia of visceral peritoneum with early fibrinous peritonitis.

On opening up the duodenum part of an *Ascaris lumbricoides* was found projecting from the ampulla of Vater into the lumen of the gut (Fig. 332). The pancreatic duct was then split up and the body of the ascaris was found passing along the duct and then turning round into the duct of Santorini, so that both ducts were effectually blocked. Bile staining of the terminal inch of the duct of Wirsung, and dilatation of the common bile-duct were present, and bile could easily be expressed from the gall bladder into the duodenum. Great congestion of the lungs. Liver fatty. Moderately fat woman. Healing varicose ulcer was found on the left calf.

PARTIAL OBSTRUCTION OF THE PANCREATIC DUCT BY ROUND WORMS

By LIEUT.-COLONEL NOVIS, I.M.S., BOMBAY

THE presence of worms in the pancreatic duct must be rare, and though these parasites often inhabit the intestine in Eastern countries, on no previous occasion have I found them blocking a duct, but have frequently observed them free in the peritoneal cavity in cases of perforation of the intestine and in one of my cases a bunch of fifty-nine worms caused acute obstruction.

B. H., a Hindu female, age 12 years, was admitted to hospital on April 6, 1922 for recurring attacks of severe abdominal pain.

The history, as is usual with Indians of the hospital class, was indefinite. Her appetite had been poor, and she had had no desire for food for some weeks. Eight days previous to admission she was suddenly seized with severe pain of a colicky nature in the abdomen, and vomited once, after two or three hours the severe symptoms subsided leaving a dull continuous pain in the epigastrium. Similar attacks occurred at irregular intervals (once or twice in forty-eight hours), but there was no further vomiting. Her health in the past had been good, and as far as could be ascertained, she had had no previous abdominal trouble.

ON ADMISSION.—The patient was thin, but her condition was fairly good. Pulse and respiration normal, temperature 99°. Abdomen no distention. The recti were rigid over an indefinite tender swelling in the epigastrium, but the rest of the abdomen was quite soft, and moved freely. Motions were regular and to all appearances normal (unfortunately no analysis was made). Urine was normal.

The patient was kept under observation for some days, during which time she had several severe attacks resembling renal or biliary colic, which had no relation to food or movement. The temperature varied from normal to 101°, but lacked the regular characters of a septic chart. Blood and x-ray examinations did nothing to further the diagnosis, except from a negative point of view.

A provisional diagnosis of pancreatitis was made. Laparotomy was performed on April 17, and a greatly enlarged pancreas exposed between the stomach and transverse colon. The stomach, gall-bladder, bile passages, and the ampulla of Vater were palpated to exclude calculus, and found normal. An incision was then made in the pancreas from head to tail, opening the pancreatic duct, from which a full-sized living round worm and a partially disintegrated one were extracted. The incision in the pancreas was united with interrupted catgut sutures and the abdomen closed, leaving a soft rubber cigarette drain down to the pancreas, which was removed after forty-eight hours.

During convalescence several round worms were passed after administration of santonin, and the patient was discharged on May 10, looking fat and well, with a healthy appetite. There was no recurrence of pain after the operation.

INTESTINAL OBSTRUCTION FROM HYDRONEPHROSIS IN A PELVIC KIDNEY

By H. TEMPLE MURSELL, O.B.E., JOHANNESBURG

The following case appears to be of sufficient rarity to justify record—

In January, 1921, a male patient, age 41, was sent to the writer by his doctor, with the following history: For the past year or more he had been suffering from attacks of intestinal obstruction, accompanied by a palpable tumour just above the bladder in the middle line, which seemed movable. The tumour became softer when the attack passed off, but never entirely disappeared. Two medical men who saw the case regarded it as

a bowel tumour causing a constriction of the lumen. These attacks became more prolonged, until one of them lasted twenty-four hours. When seen by his doctor there were typical signs of obstruction, vomit was already evil-smelling, there was much colicky pain and visible peristalsis, but the pulse was still slow and strong. As on previous occasions, an enema had immediate results, feces and flatus being passed with considerable relief. Twenty-four hours later, the symptoms recurred in an aggravated form, the face having a pinched appearance, the breath being foul, and jaundice being present. As the condition was most urgent, his doctor operated immediately at the patient's farm, many miles away from my large centre. The condition found was "a tumour, the size of a big fist, lying over the sacral promontory, and in the layers of the mesentery which was tightly stretched over it, the bowel below this being collapsed. The tumour was fluctuant, and except for hanging over the brim of the pelvis, was sessile. After packing off, the swelling was incised and the contents were found to be urine. On examining the inside of the cyst two dimples were found, but no openings therefrom could be made out. The bladder was filled up with boric lotion, but none appeared in the cyst. The lining of the cyst resembled that of the urinary bladder, and it had a distinct muscular wall apart from the mesenteric layer. It appeared, therefore, to be either a sacculation of the bladder or an enormously dilated ureter. The sac was drained by a rubber catheter and attached to the parietal peritoneum. Three days later the sac was found to drain out about 8 oz. of blood-stained urine to every 10 oz. of clear urine passed per urethram. The bladder urine contained no albumin. There was the history of a bullet wound in the region of the left kidney in 1914, making it not improbable that left ureteral stricture might have ensued. All symptoms of intestinal obstruction subsided and the patient's general condition is excellent."

So far, the history is that given by his own doctor, who, faced with a case of extraordinary difficulty, dealt with it with great judgement and under most trying conditions.

On Jan. 29, 1921, the patient was admitted to the Johannesburg Hospital under the writer's care. A Jaques catheter was still draining the median suprapubic wound and urine escaped therefrom. There was pus in the catheter urine, none in that passed per urethram. The specific gravity and the urea percentage differed in the urine from the two sources. An attempt at pyelography, by means of collargol injected through the sinus, failed owing to insufficient collargol being available. The use of sodium bromide for the purpose was not known to the writer at that time. Cystoscopy revealed no evidence of bladder diverticulum. Catheterization of the left ureter gave a free secretion of a normal urine. Methylene blue injected through this left ureteral catheter flowed back into the bladder, but was not evacuated through the catheter in the sinus. Hence the sinus, whatever its character, had no communication with the left kidney and ureter. Methylene blue by the mouth was excreted both by the urethra and by the sinus catheter. Hence the sinus could be stated to communicate with the right kidney or ureter. A ureter catheter would only pass one third of an inch on the right side, hence there was obstruction of some kind, and the right renal pelvis could not be injected from below.

OPERATION.—On Feb. 14, 1922, the writer made an oblique right lumbar incision Dr. Brebner assisting. No kidney was found in the right loin. The former anterior median incision was re-opened, and the right kidney found at the brim of the true pelvis. The loin incision was extended downwards and forwards, the kidney pushed up into it by the assistant's hand in front. A hydronephrotic kidney was removed. No true pedicle could be separated out so as to define the vessels and ureter accurately. Ligation was made through the hugely distended but flabby pelvis, and the kidney and as much as possible of the pelvis were removed.

On examination, little secreting tissue remained in the removed kidney.

The patient made an excellent recovery, and has been at work as usual on his farm since. This can reasonably be accepted as a congenitally misplaced kidney with subsequent hydronephrosis causing the unusual complication of intestinal obstruction.

Up to the last attack the renal distention was apparently intermittent, but at the time of examination and operation was probably permanent.

PELVIC HÆMATOCELE IN A MALE, UNNOTICED UNTIL INFECTED FROM THE INTESTINE

By W G SPENCER, LONDON

IN January, 1916, a private, serving in Egypt, strained himself lifting ammunition, and subsequently he noticed a left inguinal hernia. In July of the same year, in France, an explosion rendered him unconscious for two days. He was subsequently admitted to the Maudsley Hospital on account of shell-shock, afterwards he was transferred to the 4th London General Hospital, and next to a convalescent home, where, after three weeks, he was operated upon for left inguinal hernia. The prolapsed omentum was transfixed and ligatured tightly. He got up after three weeks, and was considered fit for active service. Five weeks after the operation for hernia, in November, 1916, he was seized with difficult micturition without apparent cause, and a swelling was found in the hypogastrium which was not removed by the passage of a catheter. His temperature rose to 102° . There was a tense swelling in the centre of the hypogastrium, reaching two-thirds of the distance from the pubes to the umbilicus. It was dull to percussion, could be swayed from side to side, did not bulge into the rectum, and remained unaltered when a catheter was passed, through which less than 2 oz. of urine escaped.

It was supposed that he had an abscess in the *cavum Retzi*. After making an incision and separating the recti, indurated tissue was reached but on scratching through this the bladder was opened. After suturing the walls of the bladder an opening was made just above, when a quantity of greenish-blue thick fluid escaped from a smooth-walled sac situated within the peritoneal cavity. No actual clot was met with. The cavity was drained for a few days, after which the wound healed without complications.

It is not clear whether the strain or the explosion was the cause, there is a third possibility—hemorrhage from the stump of omentum after the hernia operation, but there was no sign of omentum in the abscess. At any rate, the condition passed unnoticed until infected from the intestine.

ENDOTHELIOMA OF THE LEFT KIDNEY EXTENDING DOWN THE URETER AND PROJECTING INTO THE BLADDER REMOVAL DEATH FOUR MONTHS LATER

By W G SPENCER, LONDON

ON June 15, 1915, a woman age 45, was admitted to the Westminster Hospital complaining of hematuria and a lump in the left loin. For two years she had passed blood in the urine at irregular intervals, but in her mind this was confused with equally irregular attacks of menorrhagia. She had had pain in the left loin on and off for six months, and for a fortnight had noticed a lump there.

The left loin was filled by a firm tumour of the shape of the kidney and about three times its size. It was not tender, and had the descending colon in front of it. Through the cystoscope a cauliflower-like growth was seen projecting from the left ureteral orifice. The base of the bladder was free from induration, but through the rectum the left ureter could be traced upwards forming a cord the thickness of the finger.

On June 25 a transverse incision was made immediately above the pubes, and continued outwards parallel to Poupart's ligament. This was deepened through the abdominal wall without opening the peritoneum until the bladder wall was exposed around the end of the ureter. The bladder was then opened, and the interior found quite healthy except for a friable papilliferous growth the size of two thumbs, attached to the ureteral orifice by a narrow pedicle. A ring of bladder wall including this was cut out

and on dividing the ureter between clamps it proved to be thickened to the size of the finger by infiltration of its wall and not by the dilatation of its lumen. The abdominal incision was now carried upwards over the line of the ureter into the loin and the peritoneal cavity opened. The hand in the peritoneal cavity failed to discover any extension of the disease outside the capsule of the kidney, and the kidney and ureter were readily removed. Whilst this part of the operation took thirty-five minutes, the suturing occupied twice that period, one drain was placed in the loin, and another between the front of the broad ligament and the bladder. Healing occurred without complication, and the patient was discharged at the end of the month. She was seen once afterwards, apparently doing well, but four months after the operation she was admitted to the Infirmary in a very weak state, complaining of pain in the left loin and over the pubes. The abdominal scar was sound, and nothing was discovered on palpating the abdomen. There was no post mortem examination.

PATHOLOGICAL DESCRIPTION (*by the late Dr R. Hebb*)—The kidney tumour removed had retained approximately the shape of the kidney, and was about three times the size. On section, the pelvis and calices were found dilated and filled with whitish, semi-fluid grumous material, the solid part was formed by a vascular new growth. Microscopic sections showed large alveoli lined by columnar cells, but the cells towards the centre of the alveoli were spheroidal. Blood-vessels were seen in the walls of the alveoli. The ureter was regularly thickened and oedematous, the oedematous tissue being formed by soft myxomatous malignant infiltration, chiefly composed of small round cells of a lymphatic type. The growth in the bladder had the structure of a villous tumour, a central blood-vessel being surrounded by endothelioid cells. Dr Hebb considered the disease to be an endothelioma.

Presumably death followed infiltration by the growths of the region of the solar plexus and receptaculum chyli.

LARGE INTRAPERITONEAL (? PAROVARIAN) CYST DISAPPEARING AFTER DRAINAGE

By W. G. SPENCER, LONDON

THE patient, age 54, had suffered from indigestion all her life, but otherwise was in fairly good health up till three years previous to admission, at that time, soon after the menopause the abdomen rapidly increased in size, and at the end of one month was enormously distended. She was then admitted to the Lambeth Infirmary and the abdomen was tapped, a large quantity of fluid was withdrawn. For a short time the abdomen remained normal in size, but soon filled again. Altogether the patient was tapped four times in two months. She was then discharged from the Infirmary, and remained fairly comfortable for a period of two years.

When admitted to the hospital in June, 1916, the patient was much wasted and very weak, the abdomen was enormously distended, a rectocele of the size of two fists was protruding through the anus. On further examination, the abdomen was found to be distended with fluid which gave a marked thrill. The protrusion was most marked towards the middle line as compared with the flanks, in the epigastrium the protrusion was resonant, the rectocele was irreducible owing to tension, and on the protruding part was an ulcer the size of a five-shilling piece. The most probable diagnosis seemed to be that of a ruptured ovarian cyst.

Under general anaesthesia a small incision was made in the middle line below the umbilicus and *twenty pints* of thin brownish fluid were drawn off. The opening was then enlarged, and the hand inserted into the cavity of an enormous unilocular cyst which contained many handfuls of soft white fibrin. The whole of the cyst cavity was explored with the hand, it consisted of a thin wall which had become closely united by vascular adhesions to the parietal peritoneum and practically fused with it near the edge of the

incision The viscera could be felt through it, the uterus and ovaries were quite in place, the uterus being not at all prolapsed At the upper part of the cyst all the small intestines seemed to be bunched up in front of the pancreas, there was no enlargement of the liver or any other abdominal viscera The rectocele disappeared with the evacuation of the cyst

It was assumed from this exploration that the patient must have had a unilocular thin walled parovarian cyst, which probably prolapsed and became fixed in the pelvis, and then slowly expanded upwards, pushing the intestines before it

Two large tubes were inserted in the cyst cavity, and the rest of the wound was sewn up

An examination of the fluid from the cyst by Dr Hebb showed it to be highly albuminous, with an alkaline reaction No reaction for urea (hypobromite) No crystals (with nitric acid) of urea nitrate There were a deposit of pus and a few red blood corpuscles

The patient recovered well from the operation The cavity slowly obliterated, and finally healed up completely in December of the same year, 1916

Dec 2, 1921—The patient had remained well, but came to the hospital again on account of varicose veins, for which bandages were ordered Except for a small firm scar in the hypogastrium the abdomen appeared normal in all respects and she had no abdominal disturbance of any kind

TWO CASES OF RUPTURED SIGMOID COLON

By R M HANDFIELD-JONES, LONDON

The two cases published by Mr W G Spence in the October number of THE BRITISH JOURNAL OF SURGERY bring to mind two somewhat similar cases in my own experience, each of which shows points of unusual interest, though not the same in each

Case 1—A man, age 63, was admitted to St Mary's Hospital at 10 30 p.m., complaining of very intense abdominal pain He assured me that he had felt perfectly well that morning, and had never had a day's illness in his life There was not one single symptom which could in any way attract attention to the large bowel His bowels were opened regularly every morning, and he had not suffered with diarrhoea His relatives corroborated his story afterwards On this particular day—Sunday—he had a large dinner it middly, and tea at 4 30 p.m. Without the slightest warning, at 6 o'clock he was smitten with violent abdominal pain, and vomited once At 7 p.m. he had a hot bath, which for a short time relieved the pain, but this was only temporary On his admission to hospital I diagnosed the rupture of some hollow viscus He was in a very collapsed condition and still in acute pain He was given a small intrathecal injection of 5 per cent novocain but he was unable to withstand the resulting additional fall of blood pressure He was returned to bed, where he died almost immediately The autopsy revealed in annular carcinoma of the rectosigmoidal junction, and three inches above this a perforation of the colon, with widespread flooding of the peritoneal cavity with feces I think that the interest of this case is first, the entire absence of premonitory symptoms and secondly, the extraordinarily short space of time between the onset and the fatal result

Case 2—A man, age 56, had been attending his medical man for the past three years for mucous colitis during the whole of which time he had been apparently passing blood and mucus per anum For the week prior to admission he had been very constipated and had been unable to get his bowels to act That afternoon he had suddenly been seized with violent abdominal pain and had been sick He was seen at about 10 p.m., when he had a distended abdomen with marked tenderness and rigidity over the whole infra umbilical region His abdomen was opened by a median incision, and there was

found to be considerable faecal fouling of the peritoneum. The whole colon was distended, and the lower part full of firm faeces. A small annular carcinoma was found at the rectosigmoidal juncture, and at the apex of the sigmoid loop was a circular gangrenous area the size of a five-shilling piece, in the centre of this was a perforation the size of a shilling. The loop was emptied as far as possible, and a Paul's tube tied in. He did not survive the night, and the autopsy confirmed the operation findings, and in addition showed multiple diverticula in the whole length of the colon, quite as numerous in the ascending colon and caecum as in the sigmoid colon.

I believe I am quoting Sir Berkeley Moynihan correctly when I say that he teaches that diverticula in the ascending colon and caecum are exceedingly rare, and I think this case interesting for that reason. In addition, it would appear that the flooding of the peritoneum with the contents of the large intestine is a very different proposition in regard to prognosis from similar lesions of the upper part of the gastro intestinal tract.

REVIEWS AND NOTICES OF BOOKS

Surgical and Mechanical Treatment of Peripheral Nerves. By BYRON STOOKEY, A.M., M.D., Assistant in Neurology, Columbia University, New York, with a chapter on Nerve Degeneration and Regeneration by G. CAMPBELL, M.D., Professor of Anatomy in Michigan University. Large 8vo. Pp. 175, 217 illustrations (8 in colour) and 20 charts. 1922. Philadelphia and London: W. H. Saunders Co. 15s. net.

Interest in the surgery of peripheral nerves is stimulated by every war for it is only then that sufficient material can be obtained for study. The American Civil War called forth the work of Weir Mitchell, Morehouse, Keen, and the two later works of Head and Sherrin. This volume adds yet another to the books resulting from the experience gained in the late European War, and is the result of the authors' well known work on the subject. It opens with a comprehensive account of the general anatomy, human and comparative, of the nerve injuries of the Boer War made possible by every war for it is only then that sufficient material can be obtained for study. The individual nerves are very fully dealt with in each section before dealing with the surgery of those nerves.

The chapter written by Huber on degeneration and regeneration is an admirable account of the subject, and summarizes in an excellent manner our present knowledge. Nerve repair and operative technique are dealt with fully and the sections are admirably illustrated. In the section on sensory examination the following statement appears: "By this procedure Stookey (1916) demonstrated the sensory area of the musculospiral nerve on the dorsal part of the distal phalanx of the thumb, therefore attributed to the median nerve." This is incorrect. The area is correctly figured and described and an adequate review of the whole subject is presented. In the chapter on indications for operation is particularly good. The various operations on the nerve are fully described and many of the illustrations are shown of it. No account is given of the sensory loss resulting from injury, and no illustrations are shown of it. Much of the minute anatomy could be spared and no account is given of its symptoms and although in the bibliography place should be taken by a full account of the condition and diagnosis of it. Throughout the whole of the book this help given in recognizing injury are dismissed in six lines. At the end of this chapter other painful conditions following injury are given in almost complete neglect of the sensory phenomena following nerve injury detracts from its value. To neurological surgeons who are familiar with this side of the question the attention given to the motor side and the chapters on treatment make it a book to read.

The Practice of Surgery. By RUSSELL HOWARD, C.B.I., M.S., F.R.C.S., Surgeon to the London Hospital, Senior Surgeon to the Poplar Hospital, London. Third edition. Medium 8vo. Pp. 1280. 1922. London: Edward Arnold & Co. 30s. net.

That book of general surgery written by a single author has very many advantages compared with the multiple author system. There is an absence of overlapping, a balance in the proportioning of space, and a breadth of view which are sometimes difficult of attainment even by the most exacting of general editors. The present edition has been brought up to date, and its teaching in almost every respect conforms with modern views. One would have to be almost hypercritical to find fault with it is a sound book for the graduate study. For the practical surgeon it lacks the discussion of surgical principles and methods, which help the individual to choose for himself, and operative details are referred to only in general terms. The printing and illustrations are of a high order, and fairly representative conditions have been selected for depiction. The student who reads and digests this book will have a sound basic knowledge of the science and art of modern general surgery.

The Surgical Diseases of Children a Handbook for Students and Practitioners By FREDERICK C PYBUS, M.S., F.R.C.S., Assistant Surgeon, Royal Victoria Infirmary, Newcastle Demj 8vo Pp 408 + viii, with 288 illustrations 1922 London H K Lewis & Co, Ltd 18s net

This book represents the practical clinical teaching of the author in the wards of a children's hospital. We feel quite sure that the actual demonstrations, of which this is the mere book of words, were full of practical value, and the collection of illustrations, most of them photographs, is in itself most attractive.

The text, however, is disappointing because it is so elementary. Apart from the illustrations the book does not give as much information about the important surgical diseases of childhood as is to be found in a general text book. Such subjects as cleft palate, club foot, congenital dislocation of the hip, infantile paralysis, and surgical tuberculosis, about which we might naturally expect full and explicit teaching as to treatment are dealt with very much as we find them in an ordinary text book. On the other hand there is a disproportionate presentation of rare abnormalities of little surgical importance, such for example as supernumerary digits, club hand, and pseudo coxalgia. In the article on fractures, the suspension method of treating fractured femurs in children is not dealt with, whilst the treatment by plating is mentioned and illustrated.

As an elementary exposition of surgical practice, as seen in a children's hospital, the book is short, simple, and clear, and as such would be invaluable for nurses and dressers.

Cancer of the Breast and its Treatment By W SAMPSON HANDLEY, M.S., M.D., Lond., F.R.C.S. Eng., Surgeon to the Middlesex Hospital and to its Cancer Charity. Second edition. Pp 411 + viii, illustrated in colour and black and white 1922 London John Murray 30s net

It is sixteen years since Handley's *Carcinoma of the Breast* was first published, and the views then set forth have undoubtedly won widespread acceptance. By the permeation theory of dissemination the author has put the surgical treatment of carcinoma of the breast on a scientific basis. The second edition will be welcomed by all who know Mr Handley and his work. It has been thoroughly revised, the chapter on the natural process of repair in carcinoma, based on the author's Hunterian lecture on "The Natural Cure of Cancer", has been rewritten, and new chapters on radiological treatment, recurrence and its operative treatment, Paget's disease of the nipple, lymphangioplasty, and injury as a causative factor in carcinoma, have been added.

The author, whilst fully recognizing the importance of comparative experimental methods of research, still lays stress on the value of the study of pathology and morbid anatomy as a means of ascertaining a great deal in relation to cancer and its method of spread. Most of his work has been based on the microscopic study of large areas of tissue taken from the neighbourhood of carcinomatous growths, and the same method has been used in his investigation of Paget's disease and melanotic sarcoma.

The chapters on the radiological treatment of carcinoma of the breast are a valuable help to those who have to deal with this distressing disease, and come at a time when interest in this method of treatment is rapidly gaining adherents, and when definite instructions as to application are to a great extent lacking. The author says that no case of carcinoma of the breast should be treated by x rays or radium alone, except under the following conditions: refusal of operation by the patient, old and feeble patients with trophic cancers, and patients suffering from diabetes or cardiac or renal disease. He is strongly of the opinion that the radiologist should work in conjunction with the surgeon, he should see the case before operation, and note the exact position of the primary growth so that he may irradiate a circle of tissue of about 12 to 14 in in diameter with its centre at the position of the growth and not of necessity at the nipple, or the centre of the breast. Special care should be taken to irradiate the approximate position of the microscopic growing edge of the tumour, and extra exposures should be applied to the supraclavicular triangle, anterior mediastinal glands, and lateral chest wall. An x ray exposure before operation, and exposures for three months after, are advocated as a routine. Supraclavicular glands, if enlarged and hard but not fixed should be removed at the primary operation, but the author now depends more frequently on the insertion of tubes of radium at the time of the operation. He buries one tube in the supraclavicular fossa and one in each of the upper three intercostal spaces close to the edge of the sternum. In this way it is hoped to reduce the recurrences in these areas to the satisfactory figure of 5 per cent, as seen in the skin. The anterior mediastinal glands themselves are but occasionally attacked and then only when the growth is in the inner quadrant of the breast or when the pectoralis muscle is definitely infected. The only satisfactory method of applying radium is by burying it in the diseased tissues. In many cases especially of supraclavicular glandular enlargement associated with pain due to pressure on the brachial plexus, treatment by the insertion of tubes of radium will frequently relieve the pain, even if the growth be not materially arrested.

Chapter 17 deals with the indications for, and method of performance of the operation of lymphangioplasty. According to the author brawny arm occurs in one out of every six cases of cancer of the breast. It is due primarily to permeation of the lymphatics and the associated

perilymphatic fibrosis, contributing causes being compression of the axillary vein and obstruction of the lymph channels by growth of cancer in the glands. The operation of lymphangioplasty will relieve most of these cases, but is contraindicated if the patient is unable to take a general anæsthetic or if the threads will have to pass through cancerous tissues. Details of the operation and records of cases are given.

The chapter on Paget's disease of the nipple is new. The observations and conclusions of Paget, Batlin, and many others are given and discussed. The author's view of Paget's disease is that it is due to permeation of the lymphatics of the nipple by an underlying carcinoma of the breast and that it is always secondary to an underlying carcinoma, though in some cases the latter is so small that it is difficult of demonstration. The disease is not an epithelioma, nor is the surface epithelium the seat of malignant change; the changes are inflammatory and depend upon oedema caused by permeation and fibrosis of the lymphatics. There is an illustration of a large section in a case of Paget's disease, and a coloured picture of a case in a man.

The last chapter on injury is a case of carcinoma, though interesting, is not convincing. Several cases collected by Coley are shortly described. Coley himself is quoted as saying: "That a single injury may cause a carcinoma as well as a carcinoma is no longer open to speculation." A case is given in some detail where injury was established legally as the cause of a carcinoma of a breast. The author says: "If potentially carcinomatous epithelium is already present, injury may let loose this epithelium among connective tissues, and carcinoma may result."

The author's views throughout are clearly put, and his deductions definitely stated. The book is well printed and the illustrations are excellent. The new chapters add greatly to the value of the volume, which is one which can be confidently recommended to all who are interested in this subject.

Le Problème du Cancer. By WIL SEYMOUR BARNIMMER, Professor of Surgery at the New York Polytechnic Medical School and Hospital. Translated into French by Dr. H. ROCQUET, of Antwerp. Royal 8vo. Pp. 354. xxiv, with 38 illustrations and a number of diagrams. 1922. Paris. O. Doin.

This book, already known to many in its original English edition, has proved its value and the force of its appeal by now appearing in the French language. It represents in small compass a complete survey of the problems relating to malignant disease. It begins with a short historical note, which introduces a reference to the modern Institutes devoted to cancer research.

The next section considers the distribution of malignant disease throughout the vegetable and animal kingdom, together with its geographical and ethnological incidence. The main portion of the book is occupied by consideration of the ethnology, histology, experimental observations, clinical course, and treatment of cancer. It concludes with a short reference to the question of placing cases of inoperable cancer in special Homes and the education of the public concerning the problem of cancer.

There can be no doubt that the author has succeeded in bringing together into a very compact form a number of facts relating to the origin and treatment of the disease in a manner which makes reference to them very easy. The size of the work does not permit of either a full or a critical account of any one part of the subject. It will probably prove of great service as a text book for those dealing with the general subject of Public Health and kindred social problems.

Cancer, its Cause, Treatment and Prevention. By A. F. BRUND, M.D., Physician, Driffield Poor Law Infirmary. Demy 8vo. Pp. 120. 1922. London. John Bale, Sons & Danielsson. 8s. 6d. net.

THE practice in the wards of a Poor Law Infirmary is calculated to impress the importance and the hopelessness of the problem of cancer upon the mind of the observer. Dr. Brund, since 1902, has given a number of lectures dealing with the nature and causation of malignant disease, which are put together in the present volume. The author is intensely convinced of the infective and parasitic nature of cancer, and he is eloquent in urging that further attention should be given to the study of treatment, even though this should involve the examination of the claim of many new so-called specific remedies.

Les Tumeurs du Cerveau. By PHILIP VIGGO CHRISTIANSEN. French translation by M. POISSON, with Preface by PHILIP MARIE. Pp. 337, with 100 illustrations. 1921. Paris. Masson & Co. Fr. 25 net.

THE subject matter of this book is presented in the form of a series of clinical lectures, and is divided into chapters dealing successively with tumours of the motor region, occipital lobes, base of the brain, base of the skull, pituitary gland, cerebellopontine angle, and cerebellum. There is also an interesting chapter entitled "Diagnostic Incertain." The last section is devoted to surgical treatment, and there follow tables giving some details of cases operated upon, together with a certain amount of information as to the results.

The book is written from the point of view of a physician whose aim is to demonstrate the practical lessons to be derived from a clinical study of his patients, and in regards diagnosis, both of the localization and the nature of the lesions, there is much that is useful and instructive. The author lays stress upon the importance of knowing about the beginnings and the course of development of the symptoms, he emphasizes the need for the most careful neurological examination, but issues a warning against being 'too subtle' in the interpretation of the signs observed. His views upon what he terms 'encysted serous meningitis' are sound, for he recognizes that more or less localized collections of cerebrospinal fluid are not infrequently found in connection with tumours, and he rightly rejects the term 'pseudo tumour'. In this connection Prof. Pierre Marie remarks in his preface, in characteristic phrase, "Il n'y a pas de pseudo maladies, il n'y a que des erreurs de diagnostic."

From the surgical point of view the book is disappointing, and contains but little helpful material. The dangers of the two stage operation are rightly pointed out, and the fundamental importance of operating before symptoms of general pressure have appeared, or at least before they have become pronounced is fully appreciated. But whilst the author recognizes the fact that radical operations upon the left cerebral hemisphere rarely either produce or aggravate disturbances of speech, he perpetuates the erroneous belief that a simple 'decompression' on the left side is liable to cause such symptoms. Attempts to remove auditory nerve tumours by the transabyrinthine route are rightly condemned, but nothing is said about the intracapsular operation for their removal. Curiously enough, the surgery of pituitary tumours is scarcely mentioned.

The tables of cases operated upon are not very instructive. The first, though entitled "Radical Operation upon 21 Tumours in the Cerebral Hemisphere", contains but 13 cases in which the presence of a tumour was actually verified. It is remarkable that in as many as 6 of these 13 cases papilloedema was absent. One is unfortunately left in doubt as to what is meant by 'radical operation'. The second table contains details of 18 cases of tumour in the posterior fossa, of which 9 were extracerebellar, 5 intracerebellar, 2 intrapontine, and 2 cerebellar cysts.

The results as shown in these tables are not particularly encouraging, and whilst it may perhaps be thought that with tumours of the cerebral hemispheres a percentage of 35 cases in which "not only was life preserved but for a long while the patients were capable of working" is not to be despised, one can hardly agree that with tumours in the posterior fossa "20 per cent of good results should be considered extraordinarily satisfactory". The number of cases is, however, far too small to permit operative results to be fairly expressed in the form of percentages, and the fact that six different operators were employed detracts still further from the value of any conclusions to be derived from a study of the tables.

The book is one for the general physician, the general practitioner, and the student, rather than for the neurologist or surgeon, and may be regarded as a useful and instructive series of clinical lectures admirably presented.

Infections of the Hand: a Guide to the Surgical Treatment of Acute and Chronic Suppurative Processes in the Fingers, Hand and Forearm. By ALLEN B. KANAHEL, M.D., Chicago. Fourth edition, thoroughly revised. Medium 8vo. Pp. 500 + viii, with 185 illustrations. 1921. Philadelphia and New York. Lea & Febiger. \$5.50.

The subject of this book is one that commonly does not receive enough attention either in teaching or in practice, though the results following inadequate treatment of an acute infection of the hand may be disastrous to the patient and discreditable to the surgeon. Proper treatment of the condition can only be applied with a thorough understanding of the underlying problems—pathology and anatomy—which are well set out in the book before us. The work has reached its fourth edition and the previous issues have been noticed in these pages, so that it need not again be reviewed in detail. In the present issue the whole text has been revised in the light of knowledge concerning certain acute infections gained during the war, and a short chapter on the means of restoring function to a disabled hand has been added. The numerous illustrations are excellent, and add greatly to the value of the book.

An Index of Prognosis and End-results of Treatment. By Various Writers. Edited by A. RENDALL SHORT, M.D., B.S., B.Sc., F.R.C.S., Hon. Surgeon Bristol Royal Infirmary. Examiner for first F.R.C.S. Third edition, revised and enlarged. Roy. 8vo. Pp. 591 + vi. 1922. Bristol. John Wright & Sons Ltd. 42s. net.

WE are glad to see another edition of this useful book. It can still be claimed for it that it is unique. We know no other place in which the practitioner or specialist can find without laborious search the statistics and other information which will guide him in advising his patient as to the best line of treatment. This applies particularly to the surgeon. Surgery claims the larger part of this volume, perhaps because the results of operations lend themselves more to statistical study than most methods of treatment. Another great value of such a volume is that successive editions can throw a useful light on the progress of medicine. As the editor points out in the preface to this edition, there is now evidence in such diseases as epithelioma of the lip, strangulated

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herm, and intussusception, that the prognosis is better than at the time of the first edition in 1915.

The second edition of the book had only some results of war experience incorporated in it. The present edition has been thoroughly revised. There is much new matter on ophthalmology and the sections on venereal diseases and on obstetrics and gynaecology have been rewritten. In the list we should have liked to see a table of the incidence of the complications of pregnancy taken as a whole. It would be of interest to watch for a decrease in child birth in later editions. The comparison of medical and surgical treatment in exophthalmic goitre is instructive. We should like to know the evidence for the remarkable statement that audiosis and coma sometimes ensue in renal glycosuria. Patients often wish to know the prognosis in minor maladies which do not endanger life. We do not think the size of the book need be vastly increased by the inclusion of short notes on some more of these such as coryza, laryngitis, pharyngitis, whitlow and various skin diseases. We detected a few minor misprints, and we note that the volume has doubled in price since the first edition.

Anæsthetics in Practice and Theory. By J. BLONFIELD, O.B.E., M.D. (Cambridge), Senior Anæsthetist to St. George's Hospital and Lecturer on Anæsthetics to the Medical School. Royal Soc. Pp. 121. Six illustrated. 1922. London: Wm. Heinemann. 25s. net.

In this interesting volume the author has contributed a very useful addition to the literature dealing with anæsthetics. It has been well produced and the subject matter has been most carefully selected and clearly dealt with. In the introductory chapter Dr. Blomfield reviews the history of the art of administering anæsthetics, and, we are glad to see, although his work in the beginning of the nineteenth century is a pioneer, which he certainly was, although his work in the beginning of the nineteenth century has not generally received the amount of credit it deserved.

No attempt is made to dogmatize as to the manner in which anæsthetic agents produce their effects, the most commonly accepted theories are described, and the deduction is suggested that the essential factor is an alteration in the nerve cells, but no attempt is made to reach this view with the fact that single celled vegetable organisms are capable of being anæsthetized. In fact the question still remains open, and the difficulties which beset it are described in the four chapters dealing with the physiological actions of the more commonly employed drugs. Under this heading modern workers with light anæsthesia will find helpful accounts of the behaviour of reflexes under varying degrees of narcosis, and in different states of health, they will also find important references to the loss of temperature associated with the paralysis of deep narcosis. These chapters on the physiological action of anæsthetic drugs may with advantage be considered in association with the last chapter in the book, which deals with fatalities.

Two hundred and sixty pages are devoted to the details of administration of inhalation anæsthetics, giving the reader full advantage of the author's exceptional experience with the older as well as the newer methods in vogue, without undue bias but with wholesome warnings where necessary, especially in connection with the newer methods such as ether oil, rectal administration and prolonged gas and oxygen. In considering the supposed safety of this latter method Dr. Blomfield quotes a warning letter written by G. Williams in 1917, in which the following phrase occurs: "The death was absolutely unexplained for and his changed my ideas of the safety of nitrous oxide and oxygen entirely. I believe if I had given him ether the man would have been alive to day."

Anæsthetists will welcome the section dealing with the vexed and difficult question of the preliminary use of narcotic and other drugs, because Dr. Blomfield has given the matter much thought and is able to discuss the pros and cons very clearly. The summary of considerations at the end of the section should serve as a ready guide as to when preliminary drugs are indicated, and when not. Local analgesia and spinal and sacral methods receive careful treatment, with good description of their respective techniques. We consider that the author has done a good service to the profession by writing this work, and we hope and believe that it will require a wide popularity.

Traité de Chirurgie d'Urgence. By FLEURY LÉVY, Professor of Clinical Surgery in the Medical Faculty, Paris. Eighth edition. In two volumes, revised and enlarged. Royal 8vo. Pp. 1110, 1083 figures, 175 photographs and 20 plates. 1922. Paris: Masson et Cie. Fr. 90.

It is hardly necessary to give a long notice to this excellent French text book, which was published first in 1899, as its appearance in its eighth edition proves that it has been very widely read and appreciated, and, moreover, the English translation by Dickie and Ward is so well known. The last French edition was in 1913, and the present therefore includes the author's ideas of the lessons taught by the war about urgent surgery. In particular, gunshot wounds of the viscera, the treatment of lacerated and contaminated wounds of the soft parts, and the treatment of open fractures

have been revised and added to. One of the chief attractions of the book now, as before, is the careful and beautiful plates and diagrams. It is perhaps doubtful whether the representation of the hands of the operator and his assistant in so many operations adds to or detracts from the value of the pictures, and in any case the naked fingers placed in open wounds, especially in those of joints, is not in conformity with up to date technique.

In the section on fractures we think that undue prominence is given to complicated plaster of Paris methods and also to details of wire suturing, whilst both plating and pegging are dealt with very inadequately.

But these criticisms affect only minor portions of the work, which as a whole is beyond praise for its careful description and illustration of the problems of urgent surgery. The new English edition is shortly to be issued in one volume.

Jahresbericht über die gesamte Chirurgie und ihre Grenzgebiete for the year 1920. By PROF. DR. CARL FRANZ, Berlin. Large 8vo. Paper covers. Pp. 886. 1922. Munich. J. F. Bergmann. Berlin. Julius Springer. 53s., English price.

THIS annual review of the surgical literature forms a most valuable book of reference. It gives a short review of the work done in every department of surgery, written by over thirty collaborators, each section being followed by references to literature which, as far as we have been able to test it, are accurate and complete. The only criticism which we have to make is the lateness of its appearance. It deals with the surgery of 1920 and appears in 1922. A double index to subject matter and to authors makes reference very easy.

SHORT NOTES ON BOOKS

Surgical Diagnosis [Students' Synopsis Series]. By W. H. C. ROMANIS, FRCS, Assistant Surgeon to St. Thomas's Hospital. Crown 8vo. Pp. 302. 1922. London. J. & A. Churchill. 8s. 6d. net.

A SMALL text book for students has been written by Mr. Romanis on surgical diagnosis. It is short and clear, and arranged in such a way that reference to any particular point is very easy. A great deal of useful information has been crowded into a small space, and we have not found that accuracy has in any way been sacrificed.

The Clinical Examination of Surgical Cases: a Handbook for Students and Practitioners. By J. RENFREW WHITE, FRCS, Assistant Surgeon, Dunedin Hospital. Crown 8vo. Pp. 129. 1922. Dunedin, N.Z. J. Wilkie & Co.

ANOTHER small book dealing with the examination of surgical cases comes from New Zealand, and is by Mr. Renfrew White. It consists in a description of the best method of investigation of a number of critical surgical conditions, and is especially designed for the use of students beginning their ward work. Its use by the students responsible for note-taking would tend to produce a more thorough and uniform system of notes in any institution where it is adopted. It is interleaved so that the student may make notes as he goes along.

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EPONIMS

BY SIR DARCY POWER, K.B.E., LONDON

VIII. POTT'S FRACTURE

THE fact that Pott broke his leg and also wrote about fractures of the leg has led many to believe that he himself suffered from the injury which is now called 'Pott's fracture'. It is clear, from the account given by his son-in-law, Sir James Esdaile, that his accident was a compound fracture of the tibia. He led an active and useful life for many years afterwards, so that it is probable the fibula was not broken and that union took place with good alignment.

Writing in 1768, twelve years after his own accident, he says in his *Remarks on Fractures* —

"Whoever will take a view of the leg of a skeleton, will see that although the fibula be a very small and slender bone and very inconsiderable in strength when compared with the tibia, yet the support of the lower joint of that limb (the ankle) depends so much on this slender bone, that without it the body would not be upheld, nor locomotion performed without hazard of dislocation every moment. The lower extremity of this bone, which descends considerably below that end of the tibia, is by strong and elastic ligaments firmly connected with the last-named bone, and with the astragalus or that bone of the tarsus which is principally concerned in forming the joint of the ankle. This lower extremity of the fibula has, in its posterior part, a superficial sulcus for the lodgement and passage of the tendons of the peronei muscles, which are here tied down by strong ligamentous capsulae, and have their action so determined from this point or angle that the smallest degree of variation from it, in consequence of external force, must necessarily have considerable effect on the motions they are designed to execute, and consequently distort the foot. Let it also be considered, that upon the due and natural state of the joint of the ankle that is, upon the exact and proper disposition of the tibia and fibula both with regard to each other and to the astragalus, depend the just disposition and proper action of several other muscles of the foot and toes — such as the *gastrocnemii*, the *tibialis anticus*, and *posticus*, the *flexor pollicis longus* and the *flexor digitorum pedis longus*, as must appear demonstrably to any man who will first dissect, and then attentively consider these parts.

"If the tibia and fibula be both broken, they are both generally displaced in such manner, that the inferior extremity, or that connected with the foot, is drawn under that part of the fractured bone which is connected with the knee, making by this means a deformed, unequal tumefaction in the fractured part, and rendering the broken limb shorter than it ought to be, or than its fellow. And this is generally the case, let the fracture be in what part of the leg it may.

"If the tibia only be broken, and no act of violence, indiscretion, or inadvertence be committed, whether on the part of the patient or of those who conduct him, the limb

most commonly preserves its figure and length, the same thing generally happens if the fibula only be broken, in all that part of it which is superior to letter A in the annexed figure, (*Fig 333*) or in any of it between its upper extremity, and within two or three inches of its lower one

'I have already said, and it will obviously appear to every one who examines it, that the support of the body, and the due and proper use and execution of the office of the joint of the ankle, depend almost entirely on the perpendicular being of the tibia upon the

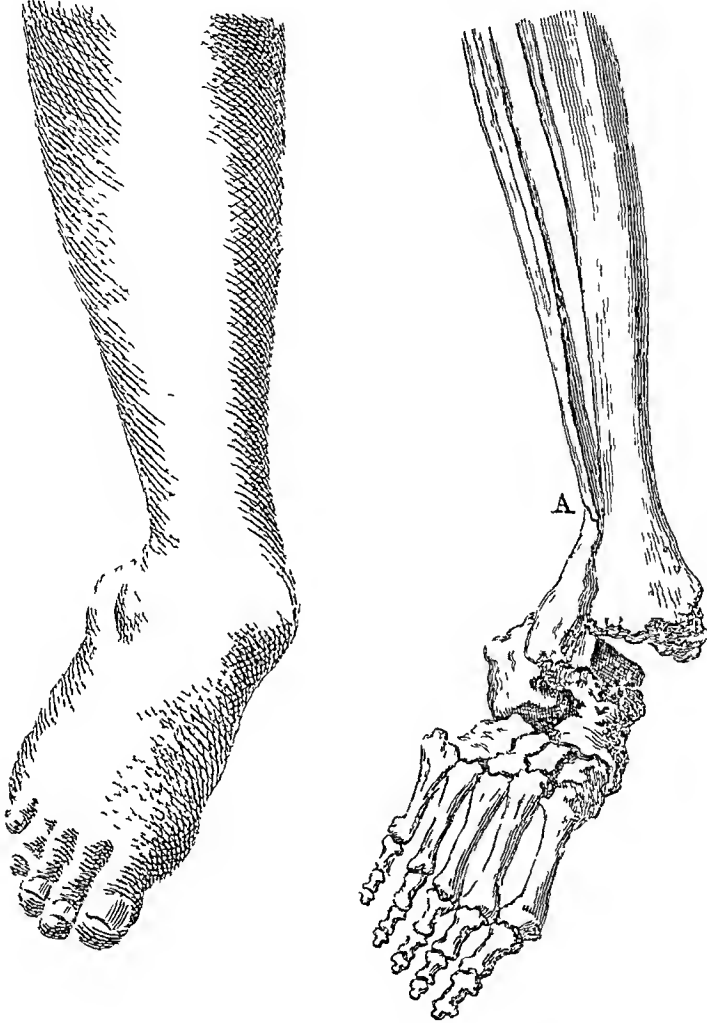


FIG. 333.—From the original illustration of Iott's fracture

astragalus, and on its firm connection with the fibula. If either of these be perverted or prevented so that the former bone is forced from its just and perpendicular position on the astragalus, or if it be separated by violence from its connection with the latter, the joint of the ankle will suffer a partial dislocation internally*, which partial dislocation cannot happen without not only a considerable extension or perhaps laceration of the bursal ligament of the joint, which is lax and weak, but a laceration of those strong tendinous ligaments, which connect the lower end of the tibia with the astragalus and

* See the figure

os calcis and which constitute in great measure the ligamentous strength of the joint of the malleoli.

This is the case when by leaping or pumping the fibula breaks in the weak part already mentioned that is within two or three inches of its lower extremity. When this happens the inferior fractured end of the fibula falls inward toward the tibia the extremity of the bone which forms the outer malleolus is turned somewhat outward and upward and the tibia having lost its proper support and not being of itself capable of steadily preserving its true perpendicular bearing is forced off from the astragalus inward, by which means the weak bursal or common ligament of the joint is violently stretched if not torn and the strong ones which fasten the tibia to the astragalus and os calcis are always lacerated thus producing at the same time a perfect fracture and a partial dislocation to which is sometimes added a wound in the integuments made by the bone at the inner malleolus. By this means and indeed is a necessary consequence all the tendons which pass behind or under or are attached to the extremities of the tibia and fibula or os calcis have their natural direction and disposition so altered that instead of performing their appointed actions they all contribute to the distortion of the foot and that by turning it outward and upward.

"When this accident is accompanied as it sometimes is with a wound of the integuments of the inner malleolus and that made by the protrusion of the bone it not infrequently ends in a fatal gangrene unless prevented by timely amputation though I have several times seen it do very well without. But in its most simple state unaccompanied with any wound it is extremely troublesome to put to rights still more so to keep it in order and unless managed with address and skill is very frequently productive both of lameness and deformity ever after.

"After what has been said a further explanation why this is so is unnecessary. Who ever will take even a cursory view of the disposition of the parts will see that it must be so. By the fracture of the fibula the dilatation of the bursal ligament of the joint and the rupture of those which should tie the end of the fibula firmly to the astragalus and the foot becomes distorted, by this distortion the direction and action of all the muscles already retracted are so altered, that it becomes (in the usual way of treating this case) a difficult matter to reduce the joint and the support of the fibula being gone a more difficult one to keep it in its place after reduction. If it be attempted with compress and strict bandage, the consequence often is a very troublesome as well as painful ulceration of the inner ankle, which very often continues, and if the bone be not kept in its place the lameness and deformity are such as to be very fatiguing to the patient and to oblige him to wear a shoe with an iron or a lead buskin or something of that sort for a great while or perhaps for life.

"All this trouble, pain, difficulty and inconvenience are occasioned by putting and keeping the limb in such a position as necessarily puts the muscles into action or into a state of resistance, which in this case is the same. This occasions the difficulty in reduction, and the difficulty in keeping it reduced, which always accompanies such accident, outward and upward makes that deformity, which always accompanies the knee moderately bent, the muscles forming the calf of the leg, and those which pass behind the fibula and under the os calcis, are all put into a state of relaxation and non-resistance, all this difficulty and trouble do in general vanish immediately, the foot may easily be placed right, the joint reduced, and by maintaining the same disposition of the limb, every thing will in general succeed very happily, as I have many times experienced.

This account of fractures of the leg and their treatment gives a fine example of the style and methods of Percival Pott's teaching. It shows him to have been a clear and logical thinker who based his knowledge partly upon anatomy and partly upon the results of his own experience.

HYPERPLASIA OF EPITHELIAL AND CONNECTIVE TISSUES IN THE BREAST. ITS RELATION TO FIBRO-ADENOMA AND OTHER PATHOLOGICAL CONDITIONS

By SIR GEORGE LENTHAL CHEATLE, LONDON

I WANT the reader to bear in mind that, however great the prominence given in this article to the hyperplasia of the connective tissues, the epithelial changes may be predominant in all tumours where epithelial elements are concerned. Although the hyperplasia of epithelium of the breast may be the predominant factor in tumour formations, or hyperplasia of epithelial and connective tissues may be mutually correlated in that process, the morphological history of the breast should be borne in mind to this extent, that had it not been for the primary dip down of surface epithelium to form the secreting elements of the gland, the connective-tissue elements would have been absent.

Another point to which I must draw attention is this, that morphologically the epithelium lining ducts and acini is really an external tissue derived from the epiblast. To describe the epithelium of ducts and acini as 'external' seems too pedantic, and in all my description I regard the epithelium as being the most internal layer.

To clarify the following description I draw attention to *Fig 334*, which represents diagrammatically the anatomical structures constituting the ducts and acini. (A) The terminal duct, (B) and (B₁) Acini, (C) The epithelium of the duct, (D) The epithelium of the acini.

Immediately underneath the epithelium there is a single layer of longitudinally arranged unstriated muscle fibre (E), which lies on the basement membrane (F), underneath which is a layer of delicate connective tissue consisting of bipolar and stellate cells (G).

The elastica (H) is seen outside the delicate connective tissue G. This relation of the elastica is maintained in ducts and acini. A very few lobules of the gland have a fine layer of elastica surrounding them, but this is very rare. Although the elastica surrounds a great many acini, it cannot be demonstrated to surround all acini even in the normal breast. (*See Acinus B₁, Fig 334*). By a normal breast I mean the average breast of 20 years of age. Sections of whole breasts examined after the age of 35 show that many glands from this age upwards cannot really be regarded as being in the normal state.

It is important to know that, whether the elastica be continued from the duct to surround the acini or not, there is no doubt that the tissues E, F, and G of the duct are always in direct continuity with the identical tissues that surround the acini. (*See Fig 334, B₁, and also Fig 348*).

(1) Represents the unstriated muscle in the duct wall outside the elastica, which gradually gets less, and disappears when the acini are reached.

The walls of ducts and individual acini are all very closely invested by fibrous tissue (K), which forms, as it surrounds the acini, the intralobular connective tissue (K₁). This fibrous tissue which so closely invests the glandular structures appears to me to be of a more separate and specialized type than that which is diffused generally throughout the breast (L). I believe this to be true for the following reasons. It (K and K₁) varies in density where no variations occur in the general fibrous tissue, and it may remain unaffected when the general fibrous tissue of the breast appears to have undergone condensation. It degenerates where no degeneration is seen in the general fibrous tissue of the gland. When undergoing degeneration it stains differently from the surrounding fibrous tissue of the gland. It shares in a very marked degree in the hyperplasia of the elastica.

when that event occurs (Figs 350 and 351) Finally it is primarily concerned in the formation of the pericanalicular and pericanalicular variety of fibro adenoma, and also some forms of multilocular papillomata *

I divide the hyperplasia I am about to describe into three classes (I) *The hyperplasia intra elastica*, (II) *The hyperplasia elastica* (III) *The hyperplasia extra elastica* These classes have a special bearing on localized and diffused fibro adenomatous conditions, and also upon the formation of papillomata

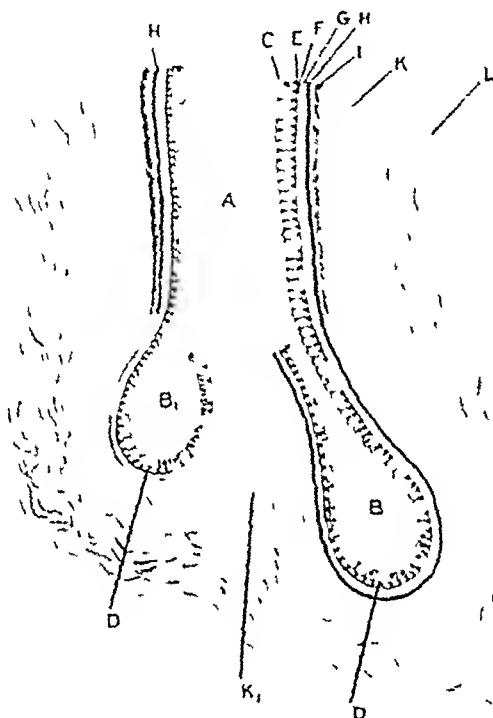


FIG. 324.—Diagram representing a duct and its acini (A) The interior of the duct (B) and (B₁) The interior of two acini (C) Epithelium (E) Small crosses representing the elastic layer of unstriated muscle fibres (F) Basement membrane (G) The delicate fibrous tissue to which great attention is drawn in this article (H) Represents the elastica to which great attention is drawn in this article (I) The unstriated muscle tissue of the duct wall (K) The fibrous tissue outside the duct wall (K₁) The intralobular fibrous tissue outside the acini K₁ is directly continuous with K To K and K₁ great attention is drawn in this article (L) Represents the general connective tissue of the breast in which all the above tissues are embedded (D) Indicates the epithelium of the acini and draws attention to the fact that the tissues of the acini (C E F G) are directly continuous with those in the duct although the elastica does not surround the acinus B₁

Class I—THE HYPERPLASIA INTRA-ELASTICA

The hyperplasia mainly occurs in the delicate connective tissue which lies immediately internal to the elastica in the ducts and acini (Figs 334, 335, 336, and 343)

The hyperplasia of the intra elastica connective tissue occurs in breasts over the age

* On reference being made to 'A Further Contribution to the Study of Cysts and Papillomata of the Breast,' BRITISH JOURNAL OF SURGERY, 1921, Vol. IX, No. 34, it will be seen that I divide papillomata into unilocular and multilocular varieties, the former a rare condition, the latter a common one which bears a very close relationship to cancer. The former arises from one stalk of fibrous tissue, the latter from many stalks composed of fibrous tissue which is either extra elastica or intra elastica in origin, and from stalks composed only of columns of epithelial cells (see Fig 338). All these forms may be seen in the same tumour

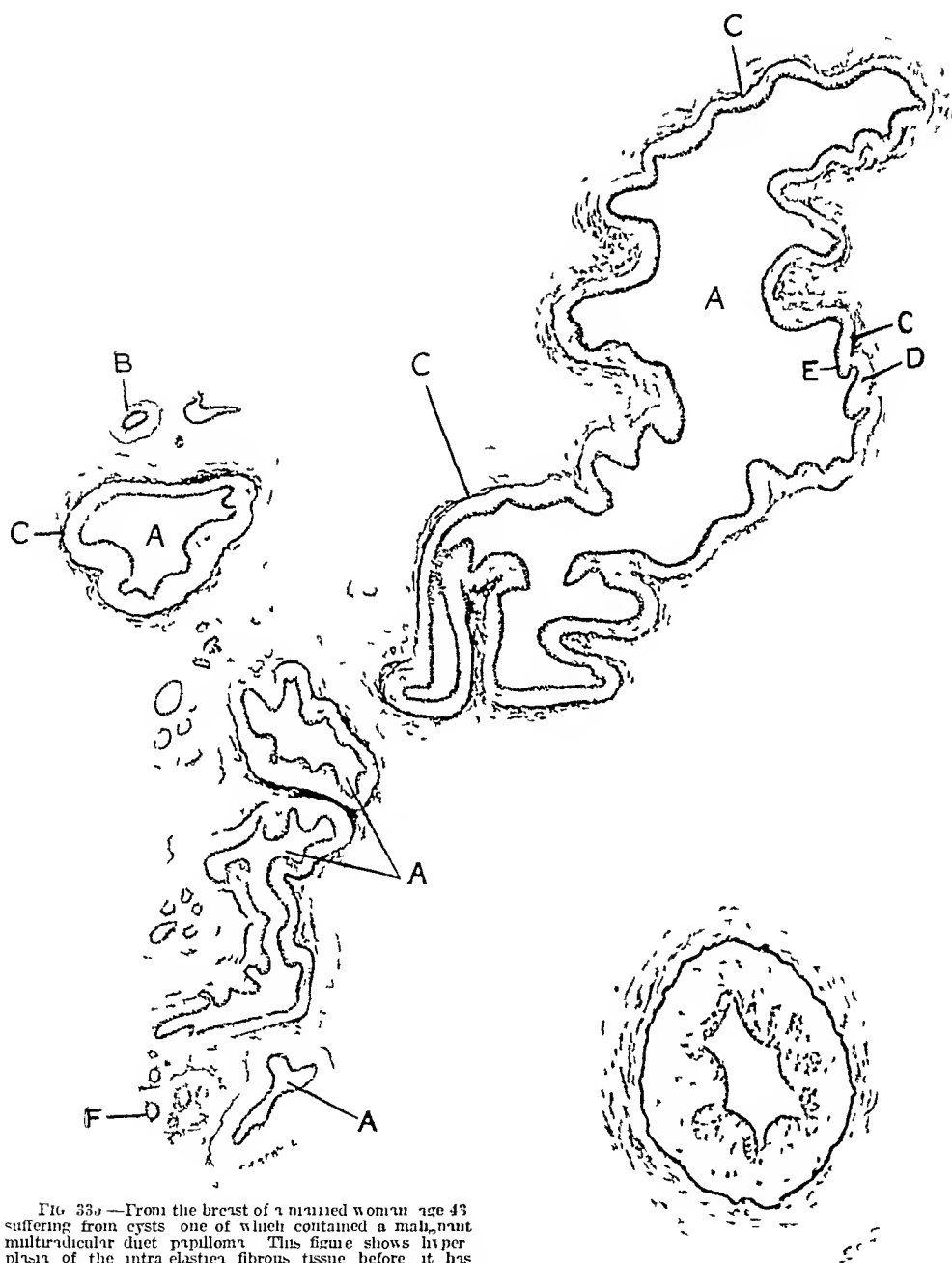


FIG. 33a.—From the breast of a married woman, age 43, suffering from cysts, one of which contained a malignant multiradicular duct papilloma. This figure shows hyperplasia of the intra-elastic fibrous tissue before it has undergone degeneration consisting of scattered bipolar and stellate cells loosely connected together by delicate fibrous tissue. (A) Ducts. (B) Acini. (C) Fibrous tissue. (D) Hyperplasia of the intra-elastic fibrous tissue. (E) The epithelium, muscle layer and basement membrane have been pushed upwards at D. (F) Acini that are not surrounded by elastica. There is no sign of inflammation in the acini and ducts affected.

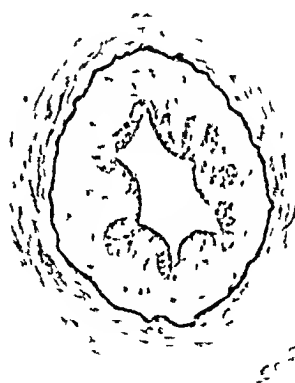


FIG. 33b.—Transverse section of a terminal duct from another part of same breast as Fig. 33a, in which the intra-elastic hyperplasia of fibrous tissue is seen in a more cellular and earlier state. (A) Elastica. (B) Intra-elastic fibrous tissue. There is no sign of inflammation in this duct.

of 30 years. It manifests itself in two ways (1) It may be *diffused*, or (2) It may be *localized* as a tumour in a duct or in *reini* respectively.



FIG. 331 - From the breast of a married woman aged 38 in which grew the intraductal tumour fibroadenoma referred to in Fig. 30.

The figure is a reproduction of a duct. (A) indicates the epithelial cell wall which in part undergoes a diffuse hyperplasia and in part some of the cells have become squamous in type. (B) The intraductal fibrous tissue which has undergone complete degeneration after hyperplasia. (C) The ductal lumen shows that the section is made at the centre of a longitudinal duct and that it is not cut obliquely.

1. In the *diffused condition* the hyperplasia affects the whole length of a duct and all or some of the *reini* from which it leads or many ducts and their attached *reini*.

The hyperplasia of this fibrous tissue can be observed best before degeneration has occurred (Figs 335, 336, and 353). The hyperplasia consists of somewhat large scattered cells some of which are bipolar and others stellate loosely bound together by delicate fibrous tissue. In these figures the complete absence of all inflammation inside or outside the glandular elements should be noted. Degeneration, which is a rule occurs after hyperplasia, may be very widespread (Fig. 337, B).

A few small isolated collections of inflammatory cells are sometimes seen in this degenerated intraelastic tissue, but only rarely, hence they appear to me to be of secondary importance. This diffused type rarely occurs in breasts in which no other lesion can be discovered. The hyperplasia may be found in some breasts in which carcinoma and multiradicular papillomata respectively or combined are present but it is of importance to note that it is often absent in breasts where carcinoma and multiradicular papillomata, respectively or combined occur. It can be seen complementing some forms of multiradicular papillomata (Fig. 338) and some forms of fibroadenomata, which belong to the hyperplasia extraelastic class (Class III, Fig. 364), where

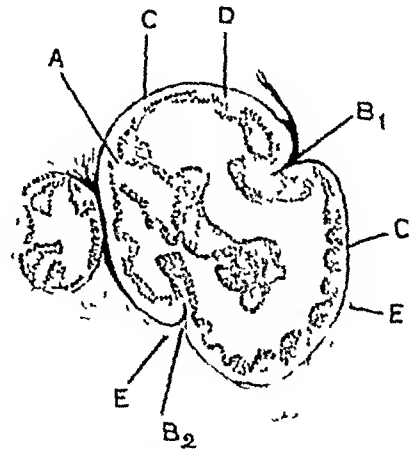


FIG. 335 - From the breast of a single woman aged 37, suffering from duct cancer. Transverse section of a duct which was situated on the internal edge of the breast. The tumour which drew attention to her condition was situated immediately opposite to the external edge. (C) The elastica. The intraelastic (D) has undergone degeneration after hyperplasia. (A) A papilloma the central stalk of which consists only of intraelastic fibrous tissue hyperplasia. This papilloma is covered by epithelium undergoing very suspicious activity. B1 and B2 are papillomata the central stalks of which contain a slight amount of elastica and extraelastic fibrous tissue (E). They belong to the hyperplasia extraelastic type, but they also show marked intraelastic hyperplasia of fibrous tissue.

it is so marked in amount that it forms the main bulk of the tumour. There are two points of interest in the diffused form of hyperplasia intra elastica. The first is the recognition of its occurrence in a much greater degree than is supposed, and the second is the fact that localized tumours may arise from it.

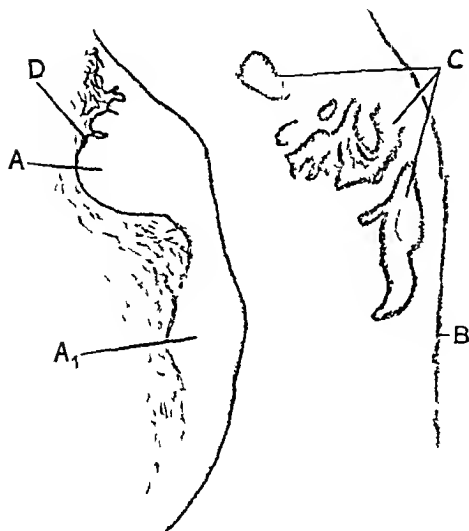


FIG 339—From the breast of a single woman, age 31, suffering from discharge of blood from nipple. Longitudinal section of an ampulla of duct (D) The elastica (A and A₁) represent respectively two oval shaped masses of degenerated intra elastica hyperplasia which bulge into the lumen of the ampulla. The epithelial, muscular and basement membrane layers have been pushed inwards. (C) Parts of a multiradicular papilloma which also grew in this ampulla. (B) The opposite wall of the ampulla represented diagrammatically.

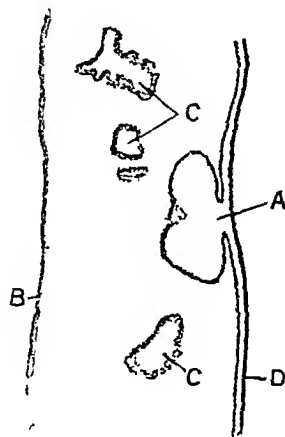


FIG 340—From the breast of a woman age 35 suffering from a discharge of blood from the nipple. Longitudinal section of ampulla of duct (D) The elastica (A) A definite pedunculated papilloma growing from the intra elastica fibrous tissue. (C) Parts of a multiradicular papilloma which grew in this ampulla. There can be seen in other sections of this extra elastica multiradicular papilloma marked hyperplasia of its intra elastica fibrous tissue. (B) The opposite wall of the ampulla represented diagrammatically.

2 The localized condition is a tumour formation which is usually superimposed on the diffused condition above described. Like the diffused form, it is usually seen in the breasts of women over 30 years of age. The tumour occurs (a) in ducts, and (b) in acini.

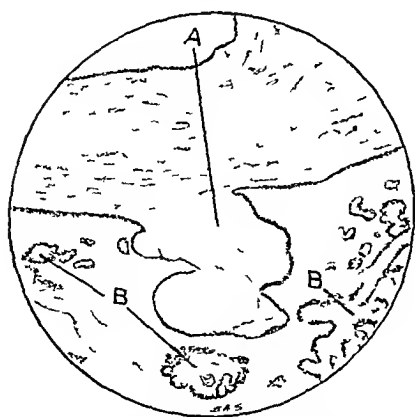


FIG 341—A papilloma similar to A in Fig 340 and taken from the same ampulla. (B) Parts of the same multiradicular papilloma seen at C in Fig 340.

a In ducts—The simplest tumour formation is shown in Fig 339, in which two oval swellings inside the elastica are placed longitudinally, and bulge into the lumen of the duct. The tissue composing them has degenerated and has pushed up the epithelial cells together with the subjacent muscle layer both of which cover their surfaces and appear to be normal. It is important to observe that the duct is also full of multiradicular papillomata. Figs 338, 340, and 341 show more complicated tumours in the definite formation of papillomata. These papillomata grew from the intra-elastica fibrous tissue of separate ducts in separate breasts. Many of the usual type of multiradicular papillomata of the duct exhibit intra elastica hyperplasia as a complication. By the "usual type of multiradicular papillomata" I mean those which possess a

connective-tissue stalk composed of extra- and intra-elastica connective tissues (Figs 338, B, and B₁.)

From the intra-elastic fibrous tissue may develop large intracanalicular fibro-adenomata. The tumour from which I describe this type of fibro-adenoma grew in the breast of a woman of 51 years (*Fig 342*). It is a true intracanalicular tumour beginning and spreading in a duct (*Fig 342 A*, and compare with *Figs 335* and *337*). This tumour is remarkable in many respects. It clearly began in the form of sessile growths springing from the intra-elastic connective tissue in the walls of a duct. The surfaces of the

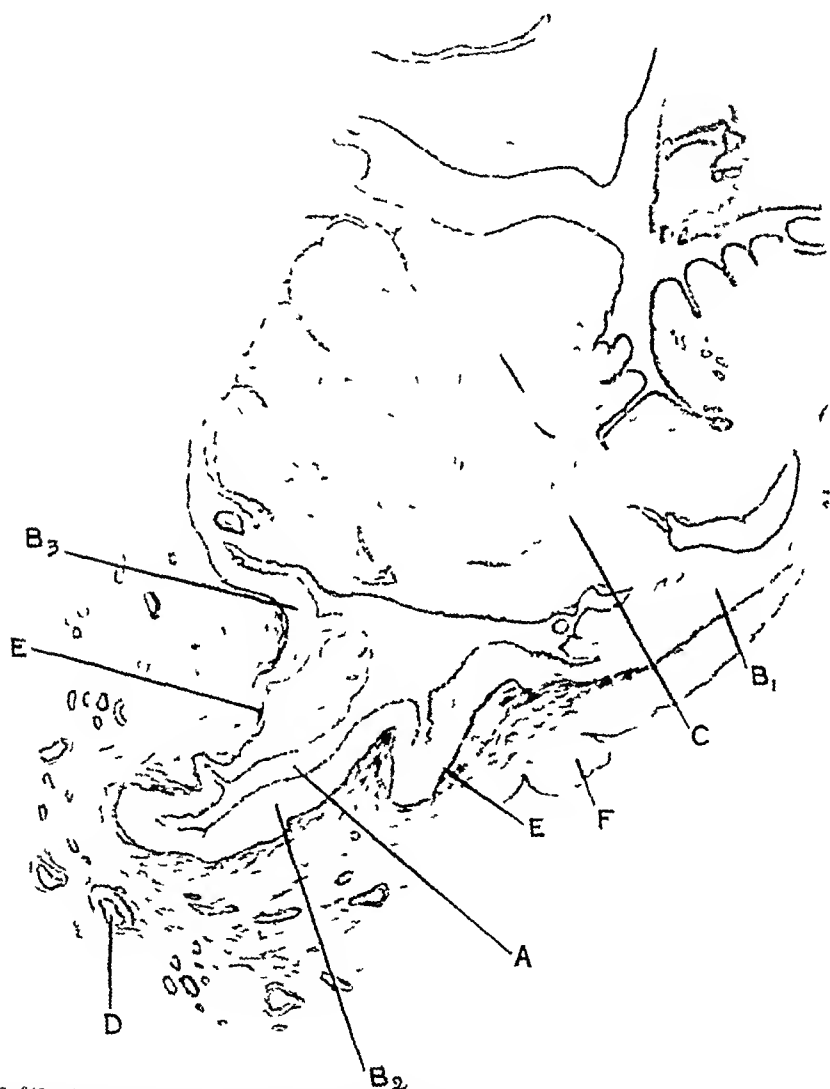


FIG 342.—An intracanalicular fibro-adenoma removed from the breast of a woman age 51. (A) Lumen of duct and from which the intracanalicular tumour (C) is growing. (B₁, B₂, B₃) Intra-elastic fibrous tissue which has undergone hyperplasia and from which the intracanalicular tumour (C) is growing. (E) The elastica. (F) A small duct cut transversely in which degeneration has occurred in the hyperplasia of the intra-elastic fibrous tissue. (F) Part of the convoluted duct A undergoing the same changes as A. There is no new formation of elastica, compare with *Fig 341*, in which it is too extensively represented.

growths are covered by the epithelial and muscle layers. At first the growths are mainly sessile, the margins showing a tendency to pedunculation. The growths may be comparatively far apart and separated, or so close together that their epithelial surfaces are in contact. Some of them become pedunculated with increase in size. The connective

tissue which forms the main bulk of the tumour is chiefly composed of bipolar and stellate cells which are loosely bound together by oedematous and delicate fibrous tissues. In other parts, denser, more fibrous spindle cells are present. A curious feature in the connective tissue of this and allied intra-aneurial tumours is the presence of scattered bundles of unstriated muscle fibres, they are not distributed universally over the tumour, but make their appearance only occasionally. I was so much astonished at their presence that I asked Dr J. A. Murray whether he agreed with me. He said he was quite satisfied they were unstriated muscle fibres.



FIG. 11.—Papilloma growing from the surface of the tumour. C (Fig. 112) from the surface of the papilloma (A) smaller and more pedunculated papillomata are shown. There is no development of elastica in this part of the tumour.

Hyperplasia of epithelium in this tumour is manifested in many remarkable ways. First, from the surfaces of these growths more or less pedunculated papillomata arise from which develop smaller and more pedunculated papillomata (Fig. 343). Further, the epithelial surface is the starting-point of other remarkable changes. Thus, it may dip down into the fibrous tissue and form depressions (Fig. 344, A), perfectly normal acini are also developed connected with stunted ducts which open on the surface of the tumour (Fig. 344, B). Lastly, comparatively long ducts may dip down from the surface of

the tissue and end in a perfectly formed lobule of acini (Fig. 344, C). These are new ducts and acini similar to those seen in the healthy, vigorous breasts of young women. These newly-formed ducts always appear as terminal, and are never surrounded by connective-tissue coats (Fig. 344). Their points of origin are most irregular and haphazard in their arrangement. The newly-formed ducts and acini in this tumour are derived from duct epithelium, a fact that would seem extraordinary if it were not remembered that all the breast epithelium arises from epiblast.

Another curious feature of these newly formed epithelial structures is that the elastica may here and there develop in its normal relation to them (Fig. 344), and often tracts of unstriated muscle are visible between the elastica and the epithelial cells, in some places even intra-elastica hyperplasia can be definitely observed. Very few blood-vessels can be seen. It is difficult to understand how such large tumours can be nourished by means of so small a blood-supply. As long as the growths are within the ducts they are encysted tumours. The pressure produced by the expansion of these tumours in the process of growth destroys the duct wall. The result of this is that the connective-tissue elements of the tumours come in direct contact with the general supporting fibrous tissue, which is so pressed upon that it appears as a definite cyst wall, for which it might easily be mistaken upon casual examination. Careful observation,

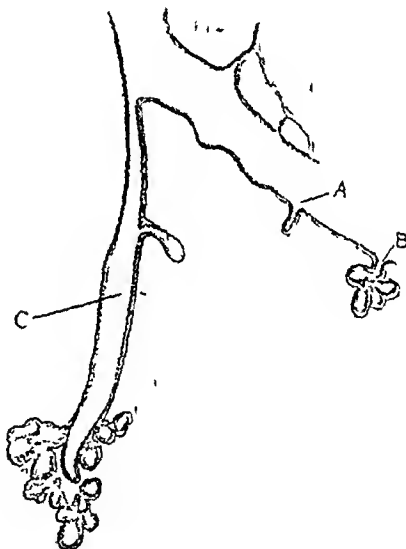


FIG. 11.—Downgrowths of surface epithelium from tumour. C (Fig. 112) (A) A depression only. (B) Stunted duct opening into acini. (C) A long duct opening into a definite lobule of acini. Elastica of new formation has developed in this part of the tumour. The elastica is drawn too diagrammatically and is not so continuous nor so marked as shown here. Its occurrence in the tumour is exceptional. (See Fig. 112, C.) It will be noted that the ducts have no connective tissue walls other than the connective tissue of the tumour. The acini are of the vigorous appearance seen in a young breast. The bundles of unstriated muscles scattered among the connective tissue of the tumour and described in the text, are not shown.

however through serial sections demonstrates that the compressed fibrous and even elastic tissues are continuous with definite strands which are lost in the structure of the

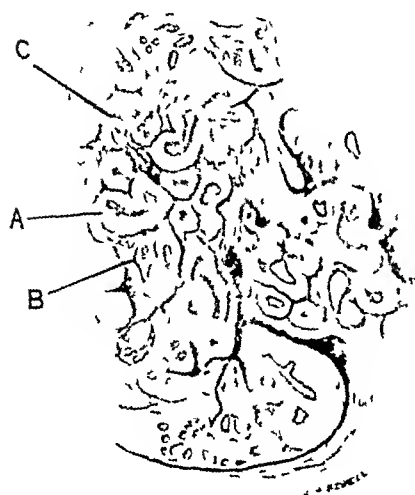


FIG 345—From the breast of a married woman, age 43, suffering from cancer. An isolated collection of acini in which the hyperplastic intra-elastic has under one degeneration. This is a small fibro-adenoma. (A) Intra-elastic hyperplasia of fibrous tissue in acini some of which are surrounded by elastica (B). The intra-lobular connective tissue is seen at C. Some acini have no elastica around them.

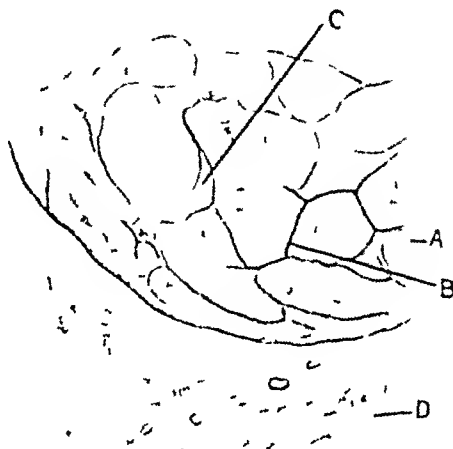


FIG 346—From the breast of a married woman, age 43, suffering from cancer. A collection of acini in the margin of a circular fibro-adenoma the whole of which is composed of the same tissue changes as here shown. In a more advanced degree it shows the same structure as seen in Fig 345. (A) The acini containing the hyperplastic intra-elastic. (B) The elastica. (C) The remnant of the intra-lobular connective tissue. (D) Surrounding tissue.

breast and have nothing to do with duct walls. This remarkable tumour contains bundles of unstriated muscle fibres, fibrous tissue, and epithelial elements. It also shows papillomata formed upon papillomata, and newly-developed vigorous young glandular tissue although it grew in the breast of a woman 51 years of age.

In Fig 349, again, is seen an intra-lobular fibro-adenoma of a duct. The tumour resembles in composition that of Fig 342, C, and arises from the intra-elastic tissue.

I believe that many intra-lobular fibro-adenomata belong to this class, and that the extension of their growth has obliterated all trace of their origin.

Lastly, in Fig 353, primary carcinoma in a duct can be seen growing upon the hyperplastic intra-elastic which is being invaded by the malignant disease at C. I regard the invasion of the structures at C by the epithelial tumour as the earliest indication of invasion by epithelial cells I have seen in a breast, and is a sign that should be sought for in epithelial hyperplasia that is contained within duct structures.

b In acini—The intra-elastic connective tissue around the acini may undergo so marked a hyperplasia that a collection of acini thus affected resolves itself into a distinct

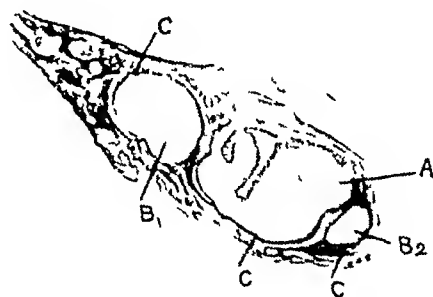


FIG 347—From the breast of a married woman, age 41, suffering from cancer. A collection of acini in the margin of a circular fibro-adenoma the whole of which is composed of the same tissue changes as here shown. In a more advanced degree it shows the same structure as seen in Fig 345. (A) The acini containing the hyperplastic intra-elastic. (B1 and B2) The remnant of the intra-lobular connective tissue. (D) Surrounding tissue.

isolated tumour and forms a fibro-adenoma (*Figs 345, 346, 347, and 348*). The intra-elastic fibrous tissue may undergo a regular form of hyperplasia round the whole acinus (*Figs 345, 346, and 348*), or the hyperplasia may be more marked at one part of the acinus and appear as an intra-acinous growth (*Fig 347*). In the examination of this tumour formation it must be remembered that where the elastica exists the tumour formation is internal to it. Where the elastica does not exist it should be realized that the tumour formation occurs in the same tissues that are directly continuous with those inside the elastica of the ducts (*Fig 343*).

Figs 348 and 349 show two reproductions from a breast kindly sent to me by Dr Creed. *Fig 348* shows very beautifully a duct, A, in which there is hyperplasia intra-elastic, terminating in a collection of acini, B, which form a part of a comparatively



FIG. 348.—From the breast of a woman age 41, suffering from cancer. (A) A) Parts of a terminal duct cut longitudinally. (B) The acini into which it leads forming part of a comparatively large fibro-adenoma of the hyperplasia intra-elastic type. (C) Elastica. There is a diffuse hyperplasia intra-elastic in the duct A.

large acinous fibro-adenoma of the hyperplasia intra-elastic type. The elastica is only occasionally present round the acini, where it occurs it is in the normal position. The acinous appearances of this part of the tumour correspond to those one would expect to see if a similar affection arose in the diagrammatic acinus B₁ in *Fig 334*. There can be no doubt that the hyperplasia intra-elastic of the duct is continuous with that of the acini A and A. *Fig 349*, are two parts of the same convoluted duct which have been cut transversely. (Compare with the same occurrence in *Fig 342, F and A*). In the two sections of the same duct are seen the beginnings of intra-acinar fibro-adenoma of the hyperplasia intra-elastic type. The pathological anatomy of these growths exactly resembles the intra-acinar tumour in *Fig 342, C*.

These tumours may appear alone, or they may complicate other forms of fibroadenoma (*Class III*). In either state they are composed of bipolar or stellate cells loosely connected by fibrous tissue, or more commonly they may be so degenerated as to exhibit little evidence of previous fibrous structure (*Figs 315 316 317*). I have no doubt then clinical signs are put down to what is called 'localized chronic mastitis' with which it has no relation.

I have no specimen in my possession which shows carcinoma arising in a remus in which there is hyperplasia intra-elastica. *Fig 373* is an example of carcinoma involving a duct thus affected.

Before leaving the subject of intra-elastica hyperplasia of connective tissue it may be well to recall that Ribbert observed the condition, and stated that he regarded it as of vast importance in the carcinoma process. I have seen this hyperplasia in breasts in which neither malignant nor benign tumours were present and also in breasts in which

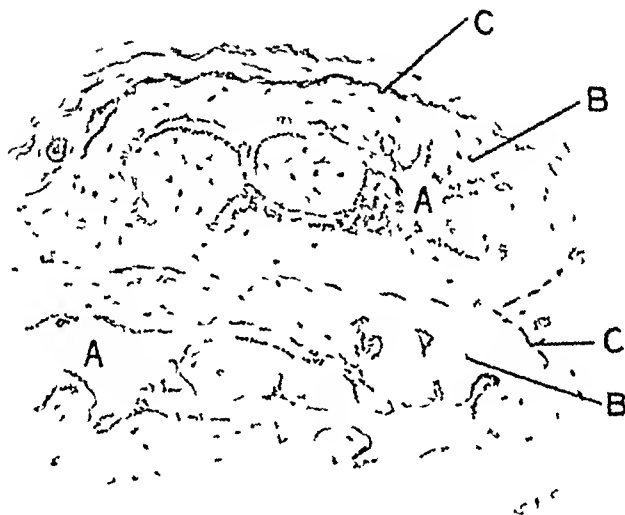


FIG. 349.—From the same breast as in *Fig 315*. Two parts of a convoluted duct cut transversely (*A A*). The duct was situated in close vicinity to the section shown in *Fig 315* in it are seen the beginnings of an intra-elastica fibro-adenoma arising from the intra-elastica (*B*) of both sections of the tube. The growth exactly resembles the structure of the tumour in *Fig 312 C*. (*C*) Elastica.

benign and malignant tumours were present in the same glands, yet I can observe nothing to contra-indicate the assumption that epithelial activity is the primary change in carcinoma. Victor Bonney, in his article on "The Connective Tissues in Carcinoma and in certain Inflammatory States that Precede its Origin", published in the *Archives of the Middlesex Hospital*, vol. vi, p. 24, shows two illustrations, Nos 21 and 22. In No 21 he depicts a duct in which the connective tissue between the epithelium and the elastica has undergone hyperplasia in a breast which he describes as suffering from 'chronic mastitis', and in No 22 he depicts a duct in the same state, but in part of a breast invaded by carcinoma. I should include the specimens from which these figures are taken under my *Class II*, the hyperplasia elastica, which occurs very frequently where there is no hyperplasia intra-elastica, and I do not believe that their relations to an 'inflammatory state' are fundamental. In the early state of hyperplasia intra-elastica inflammatory signs are absent.

Class II—THE HYPERPLASIA ELASTICA TYPE

In this class, in which there is a combined hyperplasia of the elastica and the fibrous tissue with which it is intermingled (Figs 350 and 351) the limitation of the growth is

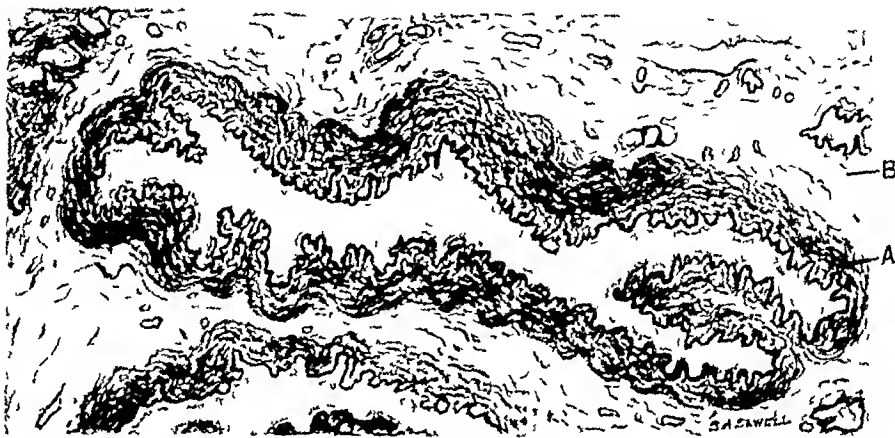


FIG 350—From a breast of a married woman age 38. The gland was removed for a painful nodular condition over the whole breast. Most of the nodules were clearly and clinically demonstrated to be irregularities of enlarged and tortuous ducts. It is diagnosed as being a procarcinoma breast (see text and footnote). The drawing shows a longitudinal section of a duct. The elastica (A) has undergone enormous hyperplasia the boundary of which is limited at the margin of the fibrous tissue which closely invests the duct (Fig 351 A). (B) The connective tissue which supports the general structure of the breast. A section stained for fibrous tissue is seen at A in Fig 351. In Figs 350 and 351 desquamative hyperplasia of epithelium is seen in the duct, and occasionally in the duct in Fig 350 slight hyperplasia in elastica can be seen.

fixed at the margin of the fibrous tissue that immediately surrounds the ducts and acini (Fig 334 K and K₁, and Fig 351 A). Most frequently the process is widely distributed,

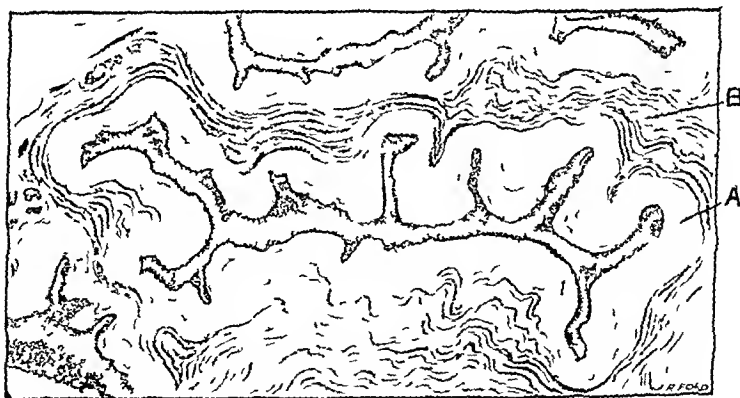


FIG 351—The same duct as in Fig 350 in the next section of the series. The elastica has not been stained. This section was stained to show the increase of fibrous tissue (A) which is intermingled with the hyperplastic elastica in Fig 350 A. (B) shows the fibrous tissue which is supporting the breast tissues in the general way. The delimitation of the fibrous tissue A from the fibrous tissue B is most marked all round the duct. The Wassermann reaction was negative.

and affects most of the ducts and acini in a breast. It is accompanied by desquamative hyperplasia of epithelium. Specimens in which I have observed it have been breasts in

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which carcinoma existed, and those which I have ventured to term 'proenimal breasts' * (Fig 352), also in breasts which suffered from Paget's disease of the nipple where ducts and acini were full of carcinoma (Fig 353). I am so impressed with these facts that I should be most careful to examine the entire breast in which there was enormous hyper-

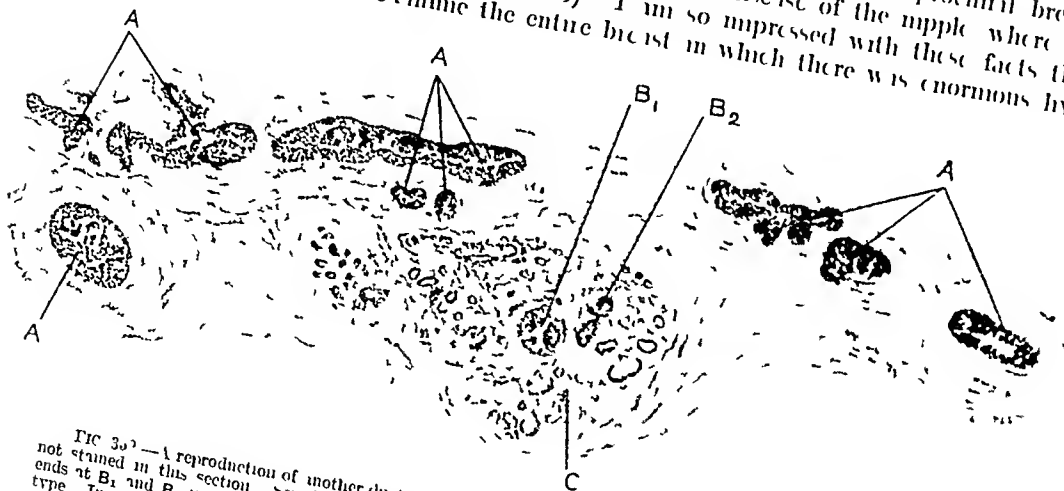


FIG 352.—A reproduction of another duct and lobule from the same breast as in Figs 350 and 351. The elastica is not stained in this section. Serial sections show that the tissue marked A all belong to one duct a branch of which ends at B₁ and B₂ in the lobule C. The hyperplasia of the epithelium seen in this duct I not descriptive in type. In my opinion it is carcinoma although the epithelial cells remain within all the wall of the duct. I am rather this type of epithelial hyperplasia renders justifiable the term 'proenimal' that I applied to the breast.

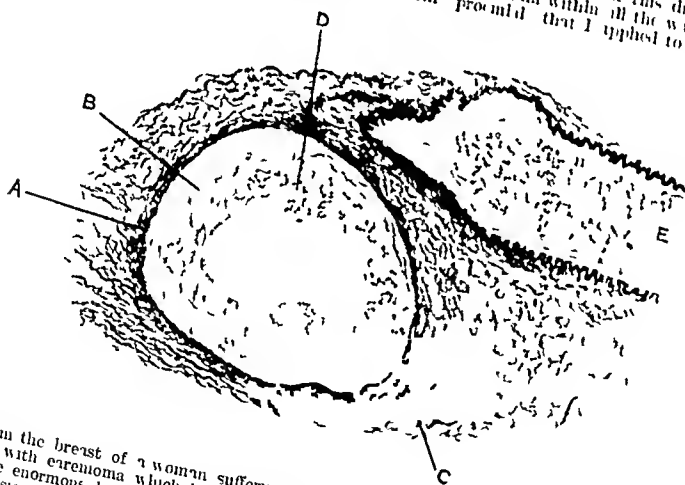


FIG 353.—From the breast of a woman suffering from Paget's disease of the nipple. One or two ducts and their acini were filled with carcinoma which had spread into the lymphatic vessels. All the ducts and most of the acini showed the same enormous hyperplasia elastica as seen in Fig 350. The figure is a transverse section of a duct (A) The hyperplasia elastica (B) Hyperplasia intra elastica which is being invaded at C by the carcinoma D full of carcinoma which has not invaded the hyperplasia elastica (E) A longitudinal branch of the same duct. It is terminates in acini and is not a blood vessel as its appearance suggests.

* The term 'proenimal breast' is employed to indicate a condition of the breast which makes it a prelude to later developments of papillomata and of duct and venous carcinomata. A proenimal breast can be recognized clinically by the presence of cysts, and in its earlier stages by the irregularity in the size of a duct or ducts. Ducts can be rendered irregular in size (1) By a desquamative hyperplasia of epithelium, (2) By a hyperplasia of epithelium which is not papillomatous or desquamative in type, but which in my opinion is malignant (Figs 352, 353), (3) By a combined hyperplasia of elastica and fibrous tissue intermingled with it (Figs 350, 351), (4) By a hyperplasia of the intra elastica fibrous tissue. These five forms of duct irregularity are often indistinguishable clinically from each other, and all are now being diagnosed as chronic mastitis. For further reference see 'Cancer of the Breast', *Brit Med Jour*, 1922 June 3.

plasia of the elastica before I passed the gland as free from carcinoma. On the other hand, there can be no doubt that most breasts which contain carcinoma do not show such enormous hyperplasia of the elastica, and the same may be said of breasts containing multiradicular papillomata. It occurs both in syphilitic patients and in patients in whom no evidence of syphilis can be obtained. In a syphilitic woman who had carcinoma in both breasts, this enormous hyperplasia of the elastica was present in both glands (*Fig 354*).

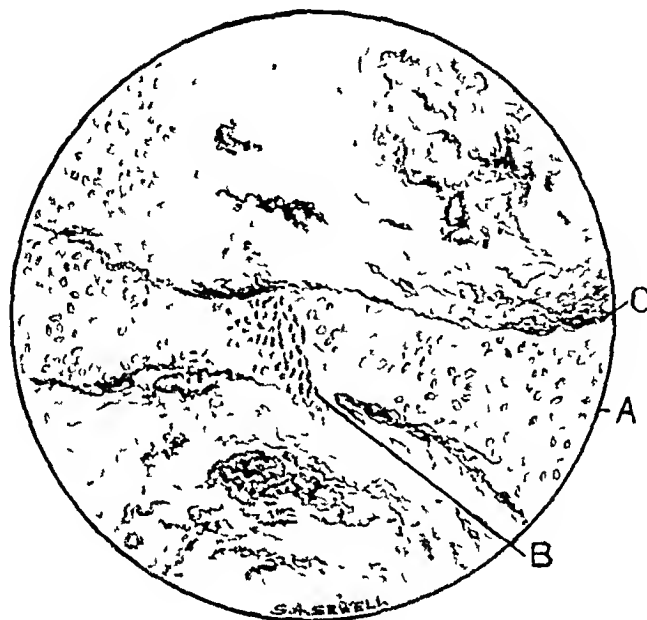


FIG 354.—From the breast of a woman, age 42 suffering from carcinoma of both breasts and a positive Wassermann reaction. The hyperplasia elastica was as great as that seen in *Fig 350* in the ducts and acini of both breasts. The figure is a reproduction of a small terminal and longitudinal duct. It contains carcinoma (A) which has invaded the hyperplastic elastica at B. (C) Hyperplastic elastica which was very irregular in distribution in this part of the terminal duct. There was no hyperplasia intra elastica in this breast, nor in the opposite breast.

The condition may be uncomplicated by the diffused hyperplasia of intra elastica fibrous tissue or may be accompanied by it to a varying degree. I can see no reason for not considering this condition a diffused form of fibro adenoma, although I have never seen it as a localized tumour. The outer coats of the arteries in these breasts show an irregular hyperplasia of their elastic fibres.

In *Fig 354* is seen an early cancer invading the elastica in a condition of this kind. It has invaded the elastica before it has penetrated the duct wall, in a section not stained to show elastic tissue this observation could not have been made.

Class III—THE HYPERPLASIA EXTRA-ELASTICA

This class concerns the fibrous tissue immediately outside the ducts, and the intra lobular connective tissue of the areola (*Fig 334*, K_1 and K), and includes mainly well known varieties of fibro adenoma of the breast.

Extra-elastica fibro adenomata are formed in the terminal segments of a breast, and affect ducts and areola. I divide this class into (1) Periecanalicular and peri acinous, which are (a) localized fibro adenomata (*Figs 355, 356, 359–367*) or (b) diffused to form a general adenomatous state of the breast and (2) Intracanalicular (*Figs 357 A, 359 C, 360 A, 364*). Except in the type of the diffused fibro adenomatosis, which occurs after 30 years, all forms may grow in young and old breasts.

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1 *Peri-acinous and Pericanalicular* —a *Localized Form* The terminal ducts and acini, as a rule, involved in this formation. The epithelial changes show themselves by a desquamative hyperplasia within the lumina, and spread in circumferential and longitudinal dimensions as the tumour grows, and although the hyperplasia of the fibrous

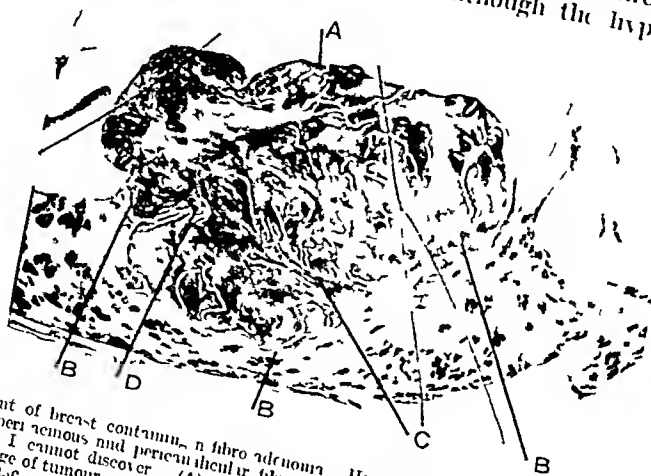
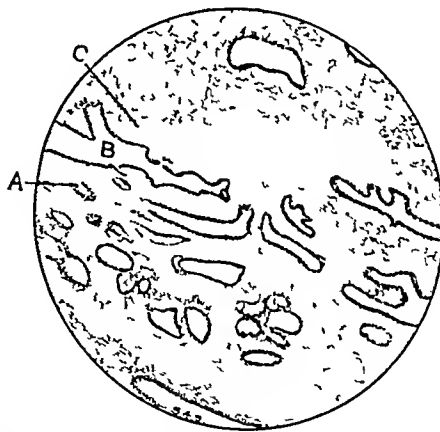


FIG 355 - Segment of breast containing a fibro adenoma. Hyperplasia extruded from fat capsule. The tumour is mainly a peri-acinous and pericanalicular fibro adenoma in which there is an intracanalicular growth the precise origin of which I cannot discover. (A) Upper edge of the tumour which impinges on fat. Here it has no capsule. (B) Lower edge of tumour round which the surrounding breast tissue appears to have grown up. (C) Portion from which Fig 356 is reproduced under higher power. (D) Portion from which Fig 357 is reproduced under

tissue manifests itself primarily in the particular parts to which I have referred (viz in the fibrous tissue that closely invests the outside of ducts and individual acini) is the tumour increases in age and size the fibrous tissue supporting adipose tissue may become affected, and the tumour may thus include fat within its boundaries (Fig 361). Mainly,

FIG 356 —From C in Fig 355. (A) A less cellular part of the specimen and continuous with the normal structure of the breast at C in Fig 355. (B) A duct the upper part of which is involved in the hyperplasia extruded from the peri-acinous and pericanalicular tumour. (C) The lower part is still in the unaffected region (A). Elsewhere in the section the peri-acinous and pericanalicular change is occurring.



however, the coarse fibrous tissue which supports the breast is pressed upon by the growth, and thereby made to appear as a capsule, but where the tumour impinges on fat there is no capsule (Fig 355, A). It is important to observe that the tumour increases in size in two ways first, by hyperplasia of the epithelial and fibrous tissue in the original tumour formation and secondly, by an exactly similar affection occurring in fresh areas of the breast. The newly affected areas may be in juxtaposition to the original tumour (Fig 366), or they may be separated from it by comparatively normal breast tissue (Fig 365), then serial sections show that the fresh area may be quite separate

from the original tumour although it may be forming in part of the same segment of the breast. The appearances in *Figs 355-363* and *367* bear testimony to this fact. The importance of this observation lies in the inevitable conclusion that this so called fibro adenoma is really a process affecting consecutive parts in a localized area of a gland that has been normal. It is wonderful to notice the sharp area of distinction that exists

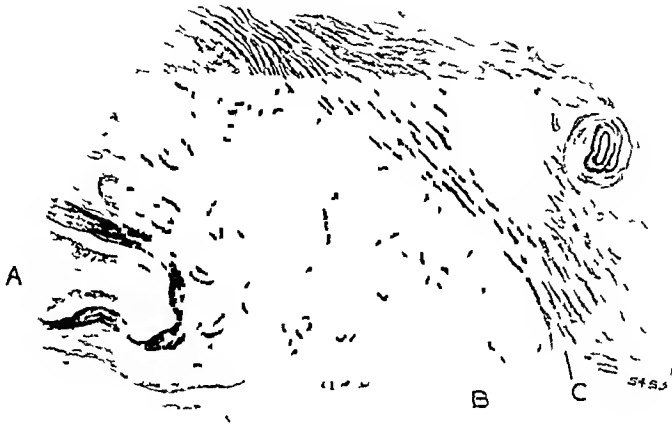


FIG 357.—Edge of tumour at D in *Fig 355*. An intracanalicular complication to the tumour has occurred at A the origin of which I cannot trace. At B a peri-acinous and pericanalicular hyperplasia of the extra-elastica fibrous tissue is occurring round glandular elements. The supporting fibrous tissue of the unaffected part of the breast is being pressed upon by the growth at C. The area B is part of the tumour.

between the hyperplasia of the fibrous tissue immediately surrounding ducts and acini (*Fig 359, B₁, B₂, B₃, B₄*) and that which forms the coarser supporting elements of the breast. In this type there does not seem to be any marked increase in the actual number of ducts and acini. These structures appear to be widely separated from one another, and do not show or suggest a multiplication of their members. This tumour forms the peri-acinous and pericanalicular fibro adenoma.

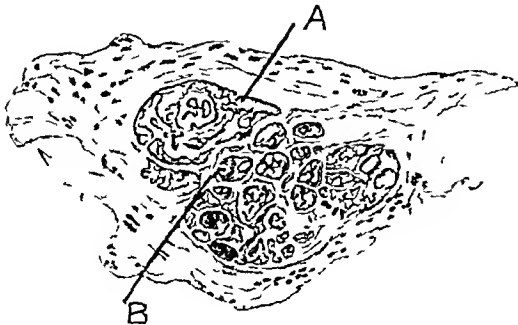


FIG 358.—A segment of a breast from a woman, age 20. It contains a fibro adenoma mainly composed of intracanalicular growth A the origin of which I cannot trace. (B) The part which is peri-acinous and pericanalicular and the tumour arises from the extra-elastica fibrous tissue round ducts and the intralobular tissue round the acini.

b A diffused form of extra-elastica hyperplasia of fibrous tissue may occur in a pericanalicular and peri-acinous form, and may affect practically the whole of a breast. The condition resembles microscopically the appearance seen in the localized condition of peri-acinous and pericanalicular fibro adenomata. Among this diffused condition can be seen an occasional small isolated tumour which exactly resembles a peri-acinous and pericanalicular fibro adenoma (*Fig 367*). The diffused fibro adenomatous condition of

breasts occurs after the age of 30, and some observers are inclined to doubt whether the small isolated tumours discovered in them are of precisely the same nature as those which

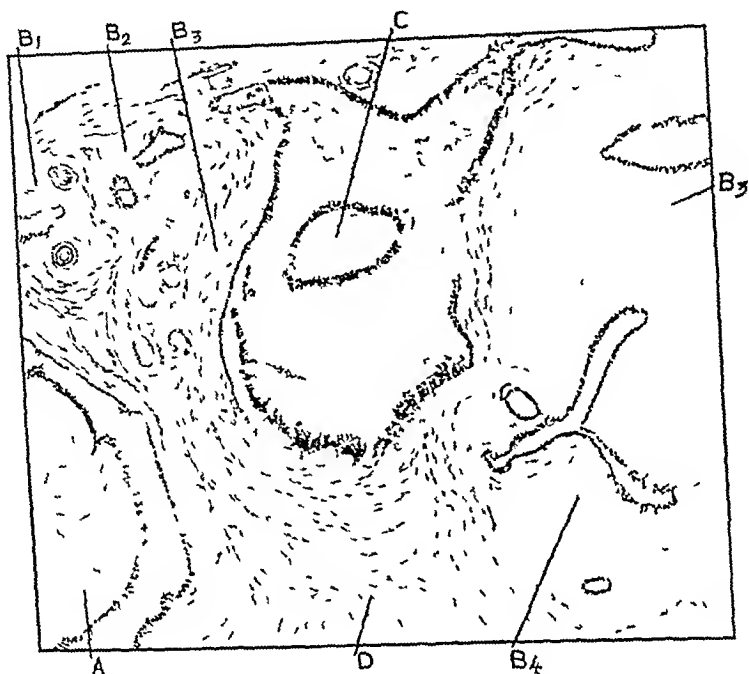
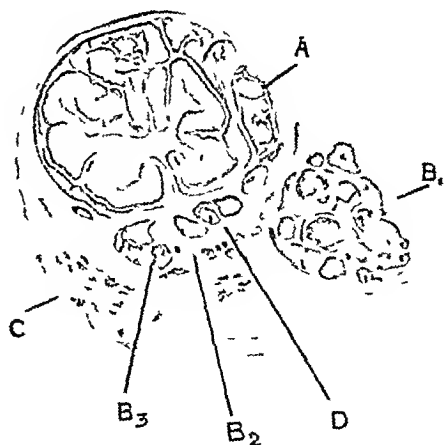


FIG 359—A reproduction under higher power of the part marked B in Fig 358. B₁, B₂, B₃, and B₄ show the periacinous and pericanalicular hyperplasia of the extralobular tissue. (D) The unaffected supporting, connective tissue of the breast. A and C are intracanalicular tumours which complicate the tumour.

occur in younger people. I can only say there is no difference between them in their growth and microscopical appearances. I believe that the diffused pericanalicular and periacinous condition I have described is a primary condition, and not merely the result of chronic inflammation.

FIG 360—Segment of a breast from a woman aged 42. It contains a large fibro adenoma that had existed for ten years. The main size of the growth is due to the large intracanalicular growth at A. B₁, B₂, B₃, and all the smaller of the deeply stained parts are composed of periacinous and pericanalicular growth of the extralobular fibrous tissue. There is no capsule to this tumour. (C) Normal breast tissue. At D there is a duct which is reproduced under higher power in Fig 361.



2 The Intracanalicular (Figs 357 A, 359 C, 360 A, and 364) —The intracanalicular type is commonly complicated by the periacinous and pericanalicular changes similar to those that occur in the localized fibro adenomata of that type. Both types commonly exist in the same tumour, there are many tumours in which the intracanalicular condition is so

pronounced that it forms the predominating part of the tumour Sessile or more or less pedunculated growths bulge into the lumina They are composed of wide central stalks

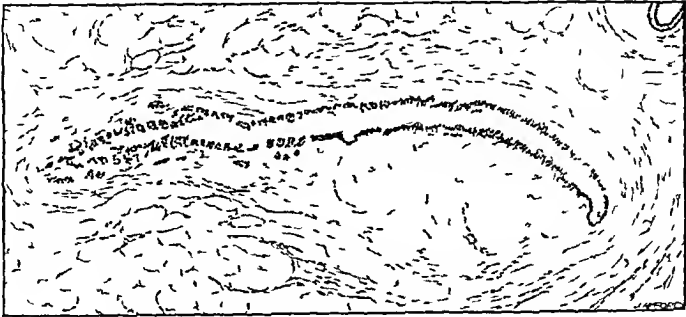


FIG 361—Section of duct from D in Fig 360 There is hyperplasia extra elastica of this duct, and there is marked hyperplasia in the fibrous tissue which is supporting the fat around the duct

of dense fibrous tissue in the centre of which the elastica is embedded, and it is because the connective tissue outside the elastica grows into the lumina that I place the tumour in the extra-elastica class This type of tumour is characterized by enormous variations in the amount of intracanalicular growths There may

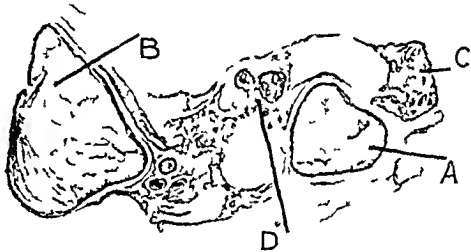


FIG 362—Portion of a breast from a woman aged 35 Three separate fibro adenomata (A, B, C) are seen They are hyperplasia extra elastica in type D marks situation of portion reproduced under higher power in Fig 363, A



FIG 363—Higher power view of Fig 362 D (A) Peri-acinous and pericanalicular hyperplasia extra elastica

be only one or two ingrowths, complicating a peri-acinous and pericanalicular tumour, or the ingrowths may be the main structure of the tumour (Fig 360) From the intra canalicular growths lateral outgrowths may arise (Fig 364) In many of these tumours very marked hyperplasia occurs in the intra elastica subepithelial tissue (Fig 364) The intra-elastica complication may be so enormous as to form the main bulk of the tumour (Fig 364) There is not the same tendency to form new ducts and acini as can be observed in the Class I intracanalicular fibro adenoma No doubt some of the intracanalicular tumours which I have been unable to classify may belong to the intra-elastica Class I type, all indications of their origin having been lost by the growth and spread of the tumours

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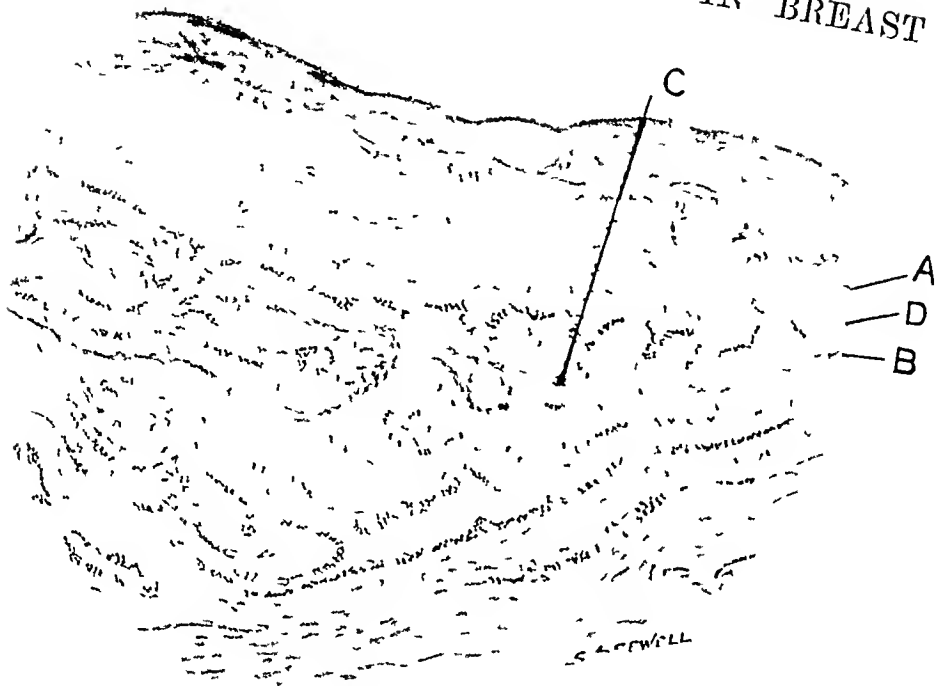


FIG 364 —Part of the fibro adenoma A in Fig 362, reproduced under higher power, showing an intraacinar growth (B) the stalk of which contains elastica (C) in the centre of connective tissue. The tumour belongs to the hyperplasia extra elastica type. Between the elastica of the stalk and the epithelium (D) hyperplasia intra elastica has occurred and forms the main bulk of this tumour. (A) A degenerative hyperplasia of the epithelium. It is difficult to say whether this tumour be duct or acinus. The amount of elastica in the stalks of the ingrowth would suggest a duct structure.

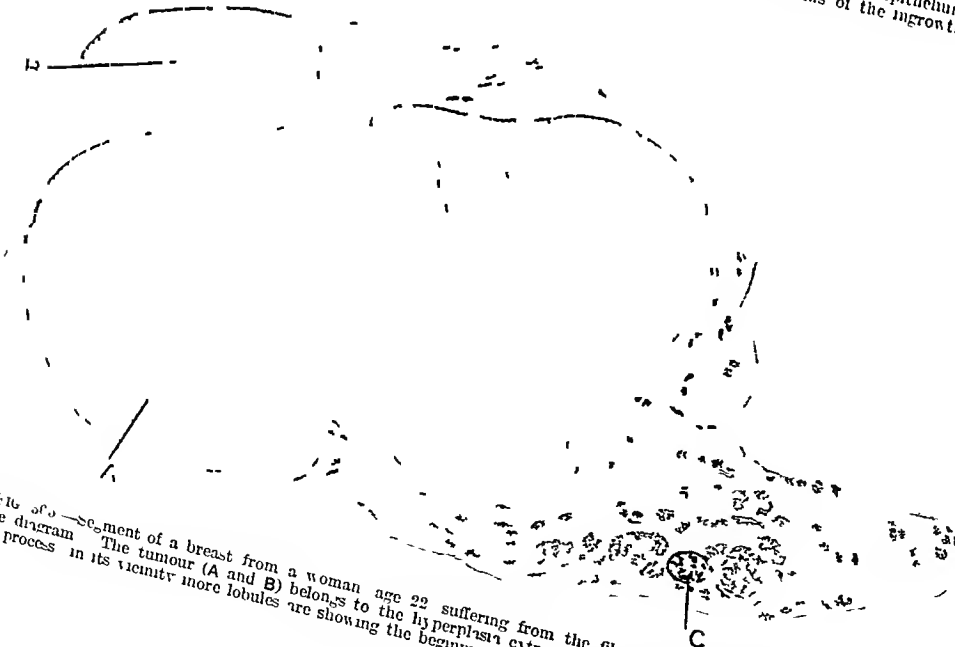


FIG 365 —Section of a breast from a woman age 22 suffering from the fibro adenoma which is represented in the diagram. The tumour (A and B) belongs to the hyperplasia extra elastica class. (C) A fresh area of the same process in its vicinity more lobules are showing the beginning of the same changes in the extra elastica tissue.

Pathological investigation of fibro-adenomata forms practically an untouched subject, and careful work in this direction is essential to isolate so many varieties. It can be inferred correctly that there is no such thing as a fibro-adenoma that does not fall into one of the three groups I have described. It can be inferred also that papillomata and intracystic growths of the breast fall naturally into the same classification.

In this paper I have described three distinct classes into which many diverse pathological changes naturally fall. I wish to impress upon the reader the fact that one class may be complicated by the presence of some pathological changes which I have placed in one of the other classes. The occasional overlapping of one class by another occurs.

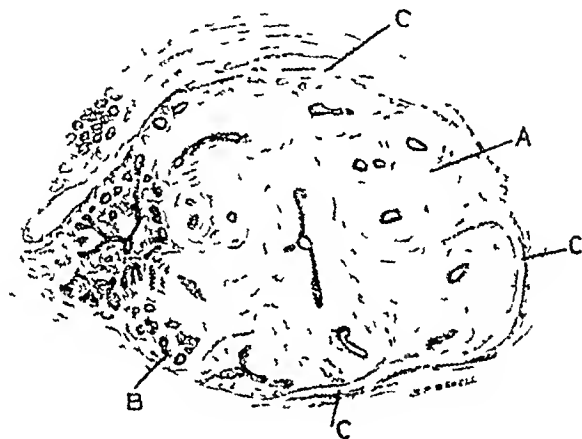


FIG. 367.—From the breast of a woman whose gland was removed because of the possibility of the condition around the nipple being Paget's disease. There was no Paget's disease. The small fibro-adenoma A belongs to the hyperplasia extra-elastica class, and is in a typical manner affecting only the intralobular connective tissue. The supporting connective tissue of the breast at C is not taking active part in the changes. In the lobule B the periacinous and pericanalicular changes seen at A are beginning.

so definitely that the trend of my observations is as follows. All the pathological changes to which I have drawn attention (and I include carcinoma) may be phases of a consecutive evolution of disease. I do not wish my classification to incur the penalty of obscuring this probable correlation, were that to happen it would destroy a notion that pervades the whole of my article.

Lastly, there is another feature in my work I wish the reader to consider. Observers recognize that all epithelium that dips down into deeper structures is not necessarily malignant, yet they do not seem to me to take into serious consideration the idea that all hyperplasia of epithelium that is confined within the ducts of the breast is not necessarily benign. I am convinced that epithelium thus situated may be malignant. The hyperplasia of epithelium in Figs 353 and 354 shows instances of what I consider to be malignant disease.

ACTINOMYCOSIS OF THE RIGHT ILIAC FOSSA

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WHILST an unbridled habit of attempting to bring about an alteration in the accepted nomenclature of a disease has nothing to commend it, I must plead a certain justification for the somewhat unusual title of this paper. The time-honoured name of ileocaecal actinomycosis has been discarded here in favour of one which, though admittedly somewhat clumsy, gives a more accurate description of the pathology of this condition. The more familiar name of ileocaecal actinomycosis undoubtedly owes its existence to the apparent but superficial similarity between this disease and that due to an infection by *B. tuberculosis*. In this latter condition the main portion of the disease is confined to the coats of the ileum, caecum, and appendix. In cases of infection in this region with *Actinomyces bovis*, investigations go to show that the intestine and appendix are themselves free from actual lesions, whilst the muscles and connective tissues of the right iliac fossa are the primary seat of the disease. Therefore, as a result of the conclusions arrived at in this paper, I have attempted to find a title which would convey a more accurate idea of the true etiology of this condition.

Invasion of the right iliac fossa with the organism of actinomycosis is not as rare a condition as some accounts might lead us to believe. A perusal of recent literature tends to give the impression that cases of this infection are becoming more frequent. A truer explanation of the apparent increase lies, I feel sure, in our greater facilities for, and our improvement in, diagnosis. Most observers agree that about 20 per cent of actinomycotic infections in man occur in the intestinal tract. In the main such lesions are located in the region of the appendix and caecum, whilst a few have been recorded as occurring primarily about the sigmoid or rectum. The stomach and small intestine appear immune or nearly so, since even in the recorded cases there is some doubt whether such lesions were really the primary seat of the disease.

The earliest clinical description of this disease in the right iliac fossa that I can find in the surgical literature of our own land was published in 1892 by Ransom in the *Transactions of the Medico-Chirurgical Society*. Like many of the recorded cases of that period, the article affords most delightful reading, being full of accurate clinical observations. This case and the post-mortem descriptions found there are referred to in a later portion of this paper. The disease in this locality, as in other parts of the body, is essentially of a chronic nature. Here, unfortunately, its course is more rapid and more fatal than in other situations in the body.

In most of the earlier cases the observers have pointed out that the disease in the ileocaecal region manifests four stages. The first with varying abdominal symptoms mainly confined to the right iliac fossa, the second with the presence of a tumour in that locality, the third with sinus and fistula formation, and the fourth with processes of repair, or more often with a gradual decline ending in dissolution.

Of recent years a change has taken place in the type of case which is reported. Observers are recording the persistence of a sinus after removal of the vermiform appendix for what was first thought to be a case of ordinary acute or subacute inflammation of that organ. Sooner or later this sinus was found to yield the sulphur granules of actinomycosis. There is, I think, an easy and reliable explanation of this apparent change. We are really dealing with two different stages in the course of identical conditions of disease. It is interesting to read in the records of Ransom's case, of the methods of treatment which were adopted even when the tumour formation was of large extent. It was evidently a

terrible thing in those days to deal surgically with even an inflammatory swelling in such a situation. At the stage of our medical knowledge then reached the disease did not come under the ken of surgeons until it was well advanced and the whole of the ileocecal region involved. On carefully perusing the early stages of the histories of these cases one is forced to the conclusion that the first symptoms were those of inflammation of the appendix, and in these days the patients would have been operated on much sooner. I feel certain this is exactly what is happening at the present time. Most of the cases recorded in recent literature describe the disease as following the removal of the cæcal appendix. The stages of this condition subsequent to appendicectomy are still identical with those of the earlier cases, which were only first seen when tumour formation was already an accomplished fact. Such evidence is sufficient to suggest that these cases, seen at an earlier stage, should be able to throw some light on the primary pathological lesions of the so-called ileocecal actinomycosis.

PATHOLOGY

Formerly, in consequence of the tumour formation and the extent of the disease when first seen, the actual anatomical site of the original focus of infection was the subject of argument and debate. It was difficult, on account of the extensive progress of the disease then existing to decide definitely whether the appendix or the cæcum was the original site of infection.

Since the appendix was generally found in the midst of the inflammatory mass it was for the most part taken for granted that this structure was the primary seat of the disease, as the following quotation from Ransom's account shows. This observer in the concluding paragraphs of his report writes: "It may be perhaps assumed that in this case the earliest seat of the disease was in the vermiform appendix, that here a fragment, or a whole grain, of corn or grass lodged, and the actinomyces parasite developed on it." This assumption was pointed out by Waring in 1905, when he published the account of seven cases of the disease in this region, and championed the view of the cæcal origin of the disease. The ascribing of the primary lesion to the appendix was at those times somewhat of the nature of a speculation, since no really minute pathological data were produced in support of such a proposition.

On the other hand, the view that such infection may at times start in the wall of the cæcum is borne out by indisputable pathological evidence.

In Chirri's case, which gave no symptoms during life, a small ulcer 1 cm. in diameter and 5 mm. thick was found post mortem in the cæcal mucosa. It was covered by a greyish layer, which in films and culture showed the presence of *Actinomyces bovis*. Agun, in 1920 Slesinger reported a case of an ulcer in the wall of the cæcum which perforated into the general peritoneal cavity. From this case also a culture of the same organism was obtained. Such early stages of this infection within the abdomen are not often met with, but they afford ample proof that the initial lesion is at times in the wall of the cæcum itself.

In the seven cases which Waring reported, the disease was of the chronic nature mostly then described. In the two post-mortem records in this observer's series, an ulcer was found in the cæcum, but in neither appendix nor ulcer could the ray fungus be demonstrated, although it was present to naked eye and microscope in the retroperitoneal tissues and liver.

In view of such absence of the organism in these ulcers one is forced to raise the question whether the ulcer was the primary lesion or secondary to the inflammatory reaction in the intestinal wall due to the extensive retrocecal disease. In the post-mortem of Ransom's case it is expressly mentioned that no lesion of the cæcal mucosa was found, neither was the organism of actinomycosis found in microscopical sections of the walls of cæcum or appendix.

It appears to me that the logical deduction from these consistently negative findings in such cases is that the ulcers in the cæcum which have been described by Waring as

the primary lesions are only of a secondary nature as far as they are concerned in the etiology of the condition

The other view, which is upheld by Kelly, Cope, and others, maintains that the lesion commences in the cæcal appendix. The possibility of this occurrence is not based on such accurately ascertained pathological facts as is that of the former view. On this subject of actinomycosis of the appendix Battle and Corner write "As this organ is so situated that it represents the back-water and sample culture tube for the cæcum, which is the first resting-place of the products of digestion after leaving the stomach, it would be surprising if actinomycosis was not found here." This statement may sound more or less true, but accurate pathological knowledge cannot be based on human expectations. More and more records are appearing in the literature, similar to the four cases recorded here, in which a sinus yielding *Actinomyces bovis* has persisted or appeared soon after an operation for acute, subacute, or chronic appendicitis.

In previous times, with the expectant treatment of appendicitis, such cases were left until tumour formation brought the sufferers under the surgeon's hand. It is on such cases that the view of the appendix being the seat of the primary lesion is largely based. Such opinion maintains that an actual lesion in the appendix mucosa was due to the ray fungus, and the infection has persisted after removal of the offending member. With this view of the exact pathological sequence of events at the onset of actinomycosis of the right iliac fossa I cannot quite agree, after a perusal of the recorded cases in the literature, and after investigating the four cases set forth at the end of this paper.

Now, wherever *Actinomyces bovis* attacks the body, whether in the cervicofacial region, thorax, or abdomen, it is of a chronic nature with its own peculiar features. There is a proliferative or neoplastic change, which is followed sooner or later by softening and pus formation in the tissues immediately surrounding the fungus. Clinically, two types have been described as occurring in the ileocecal region—the indurated or neoplastic, and the soft. Pathologically these are one and the same, it being only the time factor as regards the date of softening which differentiates them in clinical descriptions. Since the fibrous or 'woody' formation is essentially the result of a protective reaction, it is quite to be expected, as is observed, that the soft type offers a worse prognosis than does the indurated.

The most characteristic feature found in the tissues which are the seat of actinomycotic infection is the marked proliferation in the connective tissue cells before the stage of softening is reached.

When the disease has been discovered in an abscess or sinus following an appendicectomy, the case has been labelled and reported as one of actinomycosis of the appendix.

Waring, in putting forward the case for cæcal origin, has drawn a parallel comparison between this infection of the ileocecal region and an infection of the same parts with the bacillus of tuberculosis. He points out that in the latter infection the disease in the appendix is usually secondary to a primary lesion of the cæcum. It must be borne in mind, however, in making such a comparison, that the lesions peculiar to the bacillus of tuberculosis have been demonstrated in the walls of the appendix, as has also the actual presence of the specific organism of the disease. Now, in none of the recorded cases of so-called appendicular actinomycosis have the characteristic lesions produced by this infection been described as seen in the appendix itself. I can find no record of microscopic evidence of the presence of the organism of actinomycosis in the walls of the appendix. In the post-mortem records of Ransom's case, as well as in those reported by Waring this absence is noted. Gaylord and Aschoff, whilst describing the pathological histology of the appendix, definitely call attention to this fact as is seen in the following quotation from their work: "A few cases of actinomycosis have been described. The wall of the appendix in these cases shows simple inflammatory changes, the organisms being first detected in the perityphlitic accumulation of pus."

If the appendix in these cases is actually the seat of the primary lesion, it is reasonable to expect that some evidence in the tissues of the organ should be present. To a possible argument that the organism has been discharged in the process of softening in the primary

focus, I would point out that the appendix of *Case 4* in this series was submitted to an exhaustive scrutiny at a very early stage of the disease, and that the results here were also negative.

In cases where a sinus yielding sulphur granules has persisted, the appendix does not differ in appearance from appendices removed from patients who heal and recover without such complications. The four cases which are reported in this paper were all patients who presented the signs and symptoms of typical acute appendicitis of comparatively short history, the longest, of five days' duration, in *Case 4*. At the time of removal there was nothing, save in this last case, to lead one to suspect the presence of such infection. In the first three cases the appendices were opened and examined at the time of operation, but were destroyed without bacteriological or microscopic examination. They presented no signs apart from those usually seen in acutely inflamed appendices. In these cases a sinus had persisted for some time before a really energetic search was made, which resulted in the discovery of the typical sulphur granules. In *Case 4*, as the history shows, the nature of the infection was settled beyond doubt before removal of the appendix. The knowledge gained from previous experience allowed one to place a correct interpretation on the clinical findings observed at the first operation. In consequence, a careful and persistent search resulted in the discovery of the infecting organism at an early date. The appendix was therefore carefully preserved and examined in section, as was the portion of the cecal wall which was removed with it.

The appendix of this case had the appearance commonly seen in a patient who gives a history of repeated attacks which finally culminate in a reaction sufficiently acute to necessitate immediate operation. It was not markedly thickened and, on making a longitudinal section of the organ, a stricture, dating from one of the previous attacks, was present about an inch from the tip. The mucous membrane was inflamed, especially in the distal half. There was no sign of ulceration, but the portion beyond the stricture was slightly bulbous and distended with a turbid serous fluid. In this were several sulphur granules which, to the naked eye, in film, and in culture, were undoubtedly those of *Actinomyces bovis*. Although the organ was most carefully sectioned, no sign of this organism or of the characteristic tissue changes which it gives rise to could be seen in the mucous membrane or other layers of the wall of the appendix. The appearance was that of a simple inflammation with a large preponderance of eosinophil cells, which Eastwood showed is found in appendices in which inflammatory reactions have been present for several days.

Here, then, was a case in which the organism of actinomycosis was present in the lumen of the appendix, causing, as far as our pathological findings show, no trouble. Further, neither the infection nor its significant tissue changes could be found in the walls, whilst outside in the connective tissue of the retroperitoneal area, the organism was present as granules which were giving rise to the typical changes of proliferation and softening. In the post-mortem descriptions of Ransom, Waring, and of *Case 1* of this series, the main spread of the disease was in the muscles and connective tissue of the posterior abdominal wall. In some cases it has spread down into the hip joint, whilst in others, as in *Case 1*, it has caused extensive destruction of the bodies of the vertebrae. The lymphatic glands were not affected by the organism of actinomycosis, any enlargement or suppuration in these tissues was the result of the secondary infection which is invariably present in the disease in this situation.

The occurrence of pyæmia in actinomyotic infections is described as rare. In other sites of the infection this is so, though one or two cases have been reported. In the ileocecal region, however, it occurs frequently, when it is a definite portal pyæmia. Most of the post-mortems recorded describe multiple abscesses in the liver filled with the yellow granules of the ray fungus. The microscopic examination of such foci shows that they commence near the portal spaces.

In attempting to solve the question of the etiology of this condition observers have rather tended to sum up without completely weighing all the evidence. At first sight the histories and post-mortem findings of Ransom's, Waring's, and similar cases, together with

the undoubted lesions of the appendices in my four cases and in the many other recorded cases in recent literature, seem strong evidence that we must look to the appendix rather than to the cæcum for enlightenment on the early pathology of the infection of these regions with *Actinomyces bovis*. The view that the primary lesion due to the rry fungus is present in the appendix is not, I maintain, borne out by investigation. The part which is played by the vermiform appendix is a purely secondary one.

J. W. Keefe expresses the opinion that an acute or chronic inflammation of the appendix may open the door to the actinomycosis organism. Such is, I believe, the true etiology of these cases. The primary condition is that of an ordinary attack of appendicitis, in which the damage done to the walls of this organ by such inflammation has allowed the *Actinomyces bovis*, which was previously a harmless inhabitant of the lumen of the intestine, out into the retroperitoneal tissues, where it finds its best nidus in which to play havoc with the health, and oftentimes with the life, of the patient.

It is noteworthy that in all these four cases, as in others so recorded, the appendix, either wholly or for the greater portion of its course, occupied an extraperitoneal position behind the cæcum. In cases where the appendix is in the peritoneal cavity, this infection, if it occurs, first manifests itself as a mass in the wound of the anterior abdominal wall. Here it is clearly a case of wound infection at the time of operation. The fact that this condition in the right iliac fossa does not follow the cases of usual intraperitoneal position of the appendix can be explained by the extraordinary resistance shown to this infection in all cases by the peritoneal membrane. It is only in the last stages, when the patient's resistance is hopelessly broken down or when a secondary abscess in the liver has ruptured, that we see the defences of the peritoneum fall before the infection of *Actinomyces bovis*.

Such an explanation of the etiology of these cases at once raises the question of the actual character, the mode of entry, and the natural habitat of the infective agent which is responsible for actinomycosis in man. Traditions, even though not well borne out by modern knowledge, often die hard. This is true in the medical world just as in other branches of human activity. It is only in recent times that any general scepticism has arisen with regard to the theory that infection was borne by corn, barley, or grass. So much was this view held and promulgated that, if actinomycosis was found in any patient, suggestions were almost universally put to him that his habits or occupation caused him to chew raw corn or to carry straw or like material in his lips whilst at work. There is really no sound evidence for looking on actinomycotic infections as being a prerogative of rural occupations. In my own four cases there is no connection between patients and agricultural habits. In all instances they were town dwellers with work which confined them indoors. In other series of reported cases there is by no means a preponderance of rural inhabitants amongst the sufferers. There are certainly a number of cases described in which a blade of grass or an ear of corn has been found in connection with the lesion. There is, however, no actual evidence to prove that such foreign body carried the infection or that it served any function in the production of the disease other than that of producing the necessary lesion of the mucosa.

The organism which is found in such form in man, and which was discovered in the four cases of the present series, is the *Actinomyces bovis* first described by Wolff and Israel, and afterwards re-investigated and verified by Wright. There is no record to show that this organism has ever been found outside the animal body. It is an anaerobic streptothrix which will not grow on all media and which fails to reproduce itself and soon dies at room temperature. In animal inoculation experiments it gives rise to lesions containing the typical yellow granules, though it has not yet been possible to reproduce the typical 'woody' formation found in actual disease. The organism described by Bostroem, together with those obtained from grasses and corn, have always failed to give positive inoculation results in animals. In addition these latter organisms are aerobic and grow freely on all media at room temperature. On account of the cultural characteristics of his organism and those of Wolff and Israel Wright does not admit its existence outside the body, although he has no actual proof, apart from its nature in test-tube and in inoculation experiments, to substantiate this view. Lord has shown that this organism does exist in

the throat and teeth cavities of persons who exhibit no sign of the disease. In his researches typical experimental lesions were obtained in guinea-pigs.

In view of such evidence, those who maintain the source of infection to be present in corn have fallen back on a theory of the possible dual form of existence of the organism with characteristics which differ when present in the animal body from those observed when outside in the vegetable world.

At present the most we can say is that, though the life-history of this organism is but imperfectly understood, the evidence is accumulating to support Wright's view of alimentary habitation. One argument against this opinion which must certainly be considered and met in future investigations is the comparatively few cases of the disease and the entire absence of any recorded case of post-operative infection occurring in the wide realm of abdominal surgery.

DIAGNOSIS

The diagnosis of this infection can, of course, only be made with certainty after discovery of the sulphur granules of the *Actinomyces bovis* in the pus from abscess or sinus. In the cases which follow appendicectomy for an acute or subacute condition, it is well-nigh impossible to diagnose the infection at the time of operation. At this stage one would have to examine systematically the contents of every appendix removed for this specific organism. Even then the difficulties of such detection are so numerous that one cannot have enough hopes of success to justify the time expended.

The appearance of a mass in the right iliac fossa or in the actual seat of the operation should at once make the surgeon suspect the presence of this infection. It is no uncommon practice, as records show, for such a case to be explored with the fear that a swab has been left behind. An incision reveals a hard indurated mass which may or may not at this stage deliver the typical yellow granules.

In the patients who appear before us at a later stage with marked tumour formation there are certain clinical features which at least should make us strongly suspect the true nature of the condition. The patient is more usually of the male sex. The sex ratio in the four cases of this disease here reported is three males to one female, which is the proportion that Osler worked out in this infection. The patient has usually had pain of a varying degree in the right iliac fossa for some few days. He will complain of exacerbations of this pain on moving the right lower limb, especially on mounting a vehicle or on going upstairs. This feature of the pain is so characteristic of this particular infection in the ileocaecal region as to prove a strong foundation stone on which to lay an exact diagnosis. The temperature and pulse are not as a rule much raised, though these vary with the degree and virulence of the secondary infection already present.

On examining the patient the right thigh is held flexed at quite an appreciable angle, whilst any attempt to extend it, whether of a passive or of an active nature, causes excruciating pain. The abdomen moves fairly well the peritoneal cavity itself being free from infection. The main feature of the tumour is its characteristic hardness, which at times may even suggest a malignant neoplasm. Such diagnosis is as a rule put out of court by the short history of the case, the definite signs of inflammatory reaction, and the rapidity of tumour formation, with obvious involvement of the psoas muscle. One pitfall in diagnosis is to miss a primary new growth of quite small dimensions in the right testis with secondary deposits in the glands in this region. On exploration, the hard woody nature of the tissues around the appendix lying behind the cecum should make us morally sure of the true nature of the case. The ultimate confirmation of the diagnosis must wait for the finding of the sulphur granules.

It is essential for the welfare of the patient that the true nature of the infection should be proved at the earliest possible opportunity. It is often the case that the granules are not found in the primary abscess or even on several subsequent examinations. Repeated and regular searches are necessary in many cases before success crowns our efforts, since granules are only discharged in the pus when an area of softening gives way and

yields up its contents. It is often far from easy to elench the diagnosis of these cases. It requires patience and care. On one point I feel most strongly: the proper person to look for the ray fungus is the surgeon of the case or his own assistant. The easiest and most certain method is to allow the discharge from the sinus to run down the side of a sterile test-tube whilst dressing the case. If the sinus does not yield sufficient for this, gently curette the walls of the track with a spoon, and allow the blood which escapes to flow in a similar fashion. The granules will cling to the sides, allowing the liquid portion of the pus to run to the bottom of the tube. They can then be picked out and examined microscopically. If all cases were so dealt with, the diagnosis could be settled far sooner than is often the case. Too frequently the pus is kept and allowed to clot before reaching the bacteriologist. It must be remembered that it is essential for diagnosis and culture that the actual yellow granules be found and isolated. They alone bear the significant club formation. This is not easy if the pus has clotted, and the streptothrix soon dies at room temperature, so that the chances of thus finding it are greatly diminished, as experience often proves.

TREATMENT

The treatment of actinomycosis in the region of the right iliac fossa is of a more exciting and anxious nature than is the case with the same infection in other situations of the body. The condition is here complicated by the extent and variety of secondary infections which are invariably present, and also by the wide extent of the infected area when the diagnosis has been definitely proved.

The first important line of treatment with large doses of potassium iodide should, I am sure, be started as soon as the clinical features warrant even the suspicion of the true nature of the case. As regards operative measures, it is clear I think, that the condition is one in which the surgeon who holds his hand gets the best results. At first sight the prevalence of secondary abscesses in the liver of a definite pyæmic origin would, perhaps, be in favour of an extensive intestinal excision as soon as diagnosis is certain. Such an operation is bound to carry in its train a large degree of risk. Also, there is no disease found post mortem in the intestine: the infection is a retroperitoneal one and invades the portal system from behind only at a late stage of the disease. Gangolphe and Duplant, in their review of this condition, are strongly against any form of resection. The rapidly fatal result in *Case 4* is much against such treatment. Any form of resection or extensive curettage is, I am certain, strongly to be deprecated. In cases where the patient is first seen with a definite tumour formation no attempt should be made at appendicectomy. The appendix is not actually the seat of disease, and protective layers are broken down and fresh ground is thrown open to the infection by any attempt to remove it. The activities of the surgeon should be limited to ensuring free drainage when abscess formation and softening occur. Hydrogen peroxide used as an irrigating fluid is, I think the best local application. It also helps, more than any other disinfectant, to diminish the peculiarly foul odour which arises from actinomycotic infection in this neighbourhood. The smell when once recognized is peculiar to such conditions, perhaps Waring's description of an odour like sulphuretted hydrogen gives the best idea which words can convey. It is particularly offensive, penetrating, and clinging.

Medical treatment consists chiefly in the administration of increasing doses of potassium iodide until the patient is taking 100 gr three times a day. When this dose is reached many authorities advise that the drug should be discontinued periodically for a few days to allow softening to take place, and thus ensure a more certain effect of the drug on the infective organism. Some have advocated the use of γ rays in cases of this infection. This form of treatment was used in *Case 3* of this series, but with disappointing results. Heyerdahl has reported successful results from the use of radium. But here, and in the reported improvements under the application of γ rays, the lesions have all been in the cervicofacial region where one's efforts are not crippled from the start, as here, by a foul secondary infection. Some observers are high in their praises of the results of

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autogenous vaccines Here again, the same must be said of these successes as was mentioned with regard to rays and radium All four patients here reported were treated with autogenous vaccines, both for their actinomycosis and secondary infections The disappointing results of such treatment are shown only too clearly by the end-results of these cases

PROGNOSIS

The present prognosis of actinomycosis of the right iliac fossa can only be described as gloomy in the extreme The average mortality of recorded cases of abdominal infection of this nature is somewhere in the neighbourhood of 80 per cent In Warrings series 3 cases only died out of a total of 7 This mortality of 12 per cent is much lower than that of any other observer In the cases reported by Maitson the mortality-rate was 83.3 per cent The results published by Colebrook the mortality-rate was series, where the death-rate from the disease is 100 per cent The length of time which intervenes between the first symptoms and death varies from six months to two years There is no doubt that the life of the patient is of longer duration in those cases where conservative surgery has played a part It was in a large measure the main factor responsible for the lower mortality in Warrings series On the other hand, the rapid end of Case 4 in this series speaks ill for the results of any extensive surgical endeavours

Until the life-history of the infecting agent has been more fully worked out, so that measures of prevention may be adopted, the only hope of reducing the present high mortality of this disease must be in earlier diagnosis and the subsequent possibility of prompt treatment

REPORTS OF FOUR CASES OF ILEOCAECAL ACTINOMYCOSIS

- Case 1**—Miss F T Age 20 Town dweller Shop assistant
In the early part of May, 1920, the patient complained of pain in the right side of her abdomen Treated by her doctor with fomentations Pain rapidly disappeared, and patient was soon about again Some sixteen days later sudden acute attack of abdominal pain Vomited several times Temperature 100°, pulse 110 Tenderness and rigidity in right iliac fossa
May 20, 1920—Operation Appendicectomy through a para-rectal incision Appendix gangrenous and perforated lying behind the cecum surrounded with pus Drained through abdominal wound and a slab incision in loin Aug 3—Sent to convalescent home with sinus in loin Oct 16—Large abscess in loin opened and drained Nov 7—Discharged healed Dec 14—Another abscess drained through the loin A discharging sinus persisted Feb 10, 1921—Actinomycosis found in discharge from sinus Growth obtained after method of incubation described by M H Gordon Vaccine prepared Doses from 25 million to 500 million given at weekly intervals Potassium iodide administered internally July 17, 1921—Death
Post mortem findings—Body extremely emaciated Considerable post mortem discoloration of abdominal wall Several gangrenous sores on back Old operative scar over site of appendix Apertures of four old sinuses into right loin, showing marked pointing of granulations
Right lung—Collapsed, especially in upper lobes Pleura adherent at right base where pus had spread through the diaphragm from abdominal condition No secondary abscesses Left lung—Normal Heart and Pericardium—Nothing abnormal discovered beyond some fatty changes in heart muscle Spleen—Showed amyloid changes Peritoneum—Large quantity of pus present behind the peritoneum on the right side tracking up behind the right kidney and beneath right cupola of the diaphragm Pentoneum much thickened and in parts necrotic Intestines—Very adherent on right side Cecum showed no macroscopic changes Appendix absent Healthy serri in wall of cecum where stump was invaginated Right kidney—Surrounded with pus Capsule stripped easily Amyloid changes present, as was the case in the left renal organ Liver—Marked fatty changes present Advanced degree of perihepatitis Adherent to diaphragm on right side No secondary abscesses
- Case 2**—C D Age 21 Male Town dweller Clerk
Pain in the right iliac fossa for two days before admission
Aug 31, 1920—Appendicectomy through gridiron incision Appendix perforated behind the cecum Small amount of pus present Abdomen closed without drainage Sept 27—Discharged Oct 5—Tender swelling to outer side of operation scar Oct 21—Swelling explored No pus found, but a hard mass was incised and drained It lay to the outer side of and behind the cecum

Jan 25, 1921—Sinus still discharging Aug 8—Seen as out-patient with sinus still present Actinomyces found in discharge Vaccine prepared as in Case 1 Aug 11—Sinus opened up Pus containing granules evacuated Cecal wall not involved Psoas muscle widely infiltrated Treated with vaccines and potassium iodide Application of 2 rays Aug 29, 28, Sept 2, 4, Sept 5, 4, through 3 mm Al Oct 15—Discharged with persistent sinus Potassium iodide continued Developed signs of amyloid disease, and symptoms of involvement of right lung appeared later June, 1922—Death No post mortem examination

Case 3—J H Age 37 Town dweller Machinist

Three days' history of pain in right iliac fossa Vomiting one day

Oct 18, 1920—Acutely inflamed retrocecal appendix removed Wound drained Nov 8—Discharged with a sinus Dec 15—Sinus ceased discharging Jan 7, 1921—Swelling in right iliac fossa Incised A considerable quantity of pus evacuated Abscess cavity drained March 7—Actinomyces granules found in pus from sinus Treated with vaccines and potassium iodide May 3—Discharged with persistent sinus Dec, 1921—Death No post mortem examination

Case 4—T K Age 42 Town dweller Mechanic

Pain in the iliocecal region for five days Vomiting two days Right lower limb held in position of flexion for one day

March 7, 1922—Operation gridiron incision Appendix felt in a hard mass behind the cecum Abscess cavity opened and drained Appendix not removed Actinomyces was thought to be the probable infection Examination of pus obtained at operation proved negative Pus from wound examined daily March 19—Discovery of yellow granules March 20—Wound enlarged Appendix removed with one inch of cecal wall at its base All infiltrated tissue in the region of the psoas muscle removed Wound drained Treated with vaccines and potassium iodide June 3—Death Post mortem examination refused

ADDENDUM

Since writing the above, I am able through the kindness of Mr Graham Simpson, to add the notes of a further case of this condition This history well illustrates some of the more important features of these cases

May 10, 1921—Feverish cold and cough, with pain in the right iliac fossa May 23—Recurrence May 24—Temperature 99.8°, pulse 102 Rigor Tenderness and resistance over appendix Pain relieved by passage of flatus Swelling continued, but general condition improved occasional colicky pains near umbilicus, relieved by passage of motion June 3—Seen in consultation Looked well Temperature 97.6° pulse 120 Tongue clean had been vomiting all the night before Chest—Nothing abnormal, no cough Abdomen—Large, hard tumour in right iliac fossa, not very tender, quite fixed Great difficulty in extending right thigh No dysuria Appendicitis diagnosed Recommended waiting till lump had disappeared June 17—Operation appendicectomy, very large adherent appendix, no ileal kink Healed by primary union July 8—Still some thickening felt in right iliac fossa, otherwise well Nov 9—Localized tender thickening in abdominal wall under lower quarter of scum, skin over this red (? foreign body) Nov 10—Second operation incision and excision of two ounces of pus from granulation-tissue lined cavity on surface of aponeurosis, no foreign body, no cause found though carefully looked for drained Nov 14—Seen again, quite well very little pus, but no diminution of thickening Some time later actinomyces granules were found in the pus Jan 1923—Seen again Improved under potassium iodide Sinus still persists

CONCLUSIONS

1 Actinomyces of the right iliac fossa gives a clearer and more accurate description of the condition than does the older nomenclature of iliocecal actinomyces

2 The disease in this situation is more common than is generally supposed The apparent increase in its incidence can be explained by improved diagnosis

3 In the past, the large extent of the disease when first seen has masked the original site of infection In consequence, assumption rather than accurate investigation has sought to settle the point of difference of opinion between cecum and appendix

4 Cases of infection of the cecum have been reported, but no recorded case of a similar condition in the walls of the appendix can be found

5 The appendix plays purely a secondary part in the etiology of this disease of the right iliac fossa It acts as a *locus minoris resistentiae* through which the organism escapes into the retroperitoneal tissues

6 Conservative surgery gives the least unpromising results. It should be limited to simple incision, evacuation, and drainage. Resection and extensive evisceration should play no part in treatment.

7 Large doses of potassium iodide, together with an early administration of auto-genous vaccines, offer the best hope of cure.

8 Earlier diagnosis must be made if the results of treatment are to improve and if the present high mortality-rate of 80 per cent is to be reduced.

9 In all the four cases here reported the organism was similar in its cultural characteristics to that described by Wolff and Israel.

I wish to tender my best thanks to Mr. A. Cuff and Mr. Ernest Finch for kindly allowing me to use their cases, and also to Mr. S. Graham Simpson for valuable advice and help in compiling this paper.

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GIANT-CELLED TUMOURS OF TENDON ASSOCIATED WITH XANTHELASMA

By ROBERT OLLERENSHAW, MANCHESTER

In February, 1920, I saw a young Jewess, 18 years of age, who complained of a painful swelling in the region of each heel. I found, in each foot, a hard, well defined tumour situated over the tendo Achillis about two inches above its insertion. The two tumours were almost symmetrical and are well illustrated in *Fig 368*. The skin over them was a little darker than the normal skin of the part, and it was thickened from pressure.



FIG 368—Bilateral giant-celled tumours of tendo Achillis.

The patient had noticed the slow enlargement of the area for a period of three years, but had not sought any advice until she began to suffer pain from boot pressure. In all other respects the feet appeared to be normal. In the skin of the right arm, just above the inner condyle of the humerus, was a patch of what is known as xanthelasma.

At operation the following note was made: "The tendon was very greatly thickened and had many yellow-coloured areas on its surface and also infiltrating its fibres. Certain of these patches were present also in the subcutaneous tissue. The tendon was trimmed down to a little more than its accustomed size and the skin repaired."

Since that date, nearly three years ago, I have seen the girl on several occasions and, after she left the district, I have had a recent report from her medical attendant in the South of England. There has been no recurrence of the growth. A second patch of xanthelasma has appeared on the left arm in the corresponding position to the one which existed previously on the right side. The tumours are very interesting and on section, show among the tendon fibres areas where giant cells are present in considerable numbers. Figs 369 and 370 show low-power microphotographs of sections of the tumours, and Fig 371 shows a drawing made from a portion of the section seen in Fig 369 on a much larger scale. A number of tumours of similar histological characters, but arising from tendon sheaths, have been reported, and have been called sarcoma, myeloid tumour, myeloxanthoma, and various other more or less descriptive names. Targett¹ expressed the view that giant celled tumours of this type were clinically and histologically malignant in character. Bellamy² is of opinion that, although the presence of a large number of giant cells gives the tumour a claim to be called 'myeloid', yet the growth had no real right to be so classed. He regards it as due to the proliferation of the endothelial cells of the blood-vessels, and suggests the name of myeloid endothelioma. Fleissig³ describes several giant-celled tumours associated with tendon sheaths, and distinguishes them from new growths. He found them in the hand, foot, forearm, and leg below the knee, and stated as his opinion that they were not giant-celled sarcomata but inflammatory granulation growths, and should be called granulomata of tendon sheaths. Tourneur,⁴ in 1913, reviewed the literature of 93 cases of sarcomata of tendon sheaths, and found that 54 were of a xanthic type containing giant cells, 8 of these were in the lower extremity 5 being on the toes and 3 in the malleolar regions. Broders⁵ recently investigated 17 cases of this nature which have been seen in the Mayo Clinic at Rochester, USA. He found no recurrence after local removal. In the case which I am here recording the occurrence of the tumour as a bilateral condition points to some constitutional causation rather than to local new growth. The tumour was not one of the type which originates in tendon sheath, as in the cases reported by the foregoing authors. The tendo Achillis has no true sheath, and the tumour was definitely in and of the tendon itself and must have originated there. The presence of the plaques of xanthelasma in other parts of the body supports the probability of a xanthic constituent in the tumour, and the appearance of the many yellow areas infiltrating the tendon, as seen at operation, lends further support to this view. Unfortunately the block of tissue from which sections were cut was lost before any sections had been stained to show the presence

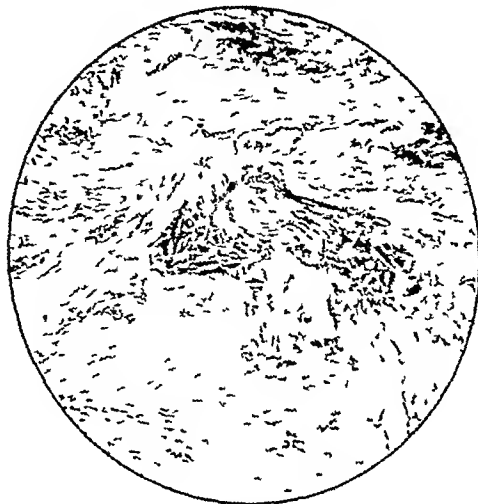


FIG 369 — Low power section of tumour



FIG 370 — Low power section of tumour

other parts of the body supports the probability of a xanthic constituent in the tumour, and the appearance of the many yellow areas infiltrating the tendon, as seen at operation, lends further support to this view. Unfortunately the block of tissue from which sections were cut was lost before any sections had been stained to show the presence

of fats There seems to be little doubt, however, that the material which occupied the spaces, shown so well in *Fig 371*, round which the giant cells are chiefly grouped, was of a lipoid character, and that the giant cells are to be regarded as being engaged in the removal of this abnormal material



FIG 371—Higher power view of part of section shown in *Fig 369*

From the clinical standpoint the question as to malignancy of tumours of this class is of great importance It appears, from the material before us, that we are thoroughly justified in regarding them as benign, having more the character of a granulomatous change than of a new growth, and that local removal is all that should be undertaken

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TUMOURS OF TENDON AND TENDON SHEATHS.*

By ST I D BUXTON, LONDON

THE subject of tumours of tendons and tendon sheaths is of interest owing to the rarity of the tumours. Observations have been made on them since 1860, and in studying the work of these observers one cannot help noticing that the greater part of it has been done by French and German surgeons and pathologists.

TUMOURS OF TENDONS

Only a few words can be said of tumours of tendons. Primary tumours are very rare and possibly do not occur at all. I can find no trace of a case reported, and in 1907 Ombredanne¹ agreed with this statement. He was not satisfied that the fibromata and sarcomata described originated in the tendon, but he acknowledged the possibility of osteomata occurring. This condition he compares to myositis ossificans. Tendons of course, are not uncommonly invaded by direct spread of malignant tumours which have commenced in the bone or connective tissue in their neighbourhood.

TUMOURS OF TENDON SHEATHS

Our attention is thus practically confined to the tendon sheath. This structure is a specialized part of connective tissue, and is therefore subject to the same neoplasms as are other connective tissues. Such neoplasms are not uncommon, therefore one searches for some reason why tendon sheaths should be so immune from tumour formation, and it is very difficult to find any facts bearing on this point.

First, our knowledge at the present time of the origin of tumour formation is extremely small.

Next, it has been found that a history of trauma is not uncommon prior to tumour formation. One has no reason to suppose that tendon sheaths are anatomically shielded from trauma, on the contrary, one would suppose that they and tendons were very liable to injury.

In the third place, in all probability chronic inflammation and irritation are predisposing factors to tumour formation. Chronic tenosynovitis is very frequent, and becoming more so with the development of athletics and machinery.

Therefore one is at a loss to account for the rarity of these tumours, especially as, although they are of small size, they cause some limitation of function of hand or foot—a sufficient reason as a rule to make pauper, peer, or physician seek the advice of a surgeon.

The classification of the primary tumours occurring on tendon sheaths is as follows—

1 *Benign*—(i) Lipoma, (ii) Fibroma, (iii) Chondroma, (iv) Ganglion.

2 *Benign, but liable to Recur Locally*, and therefore often called locally malignant—Giant celled myeloma.

3 *Malignant*—Sarcoma.

1 *Benign Tumours*—

1 *Lipomata* are uncommon, and detailed observations have been made on only eighteen cases. Two varieties are recognized. *Lipoma arborescens*² occurs, analogous in origin to those from the villi of the joints, which were first described by Muller nearly a century ago. *Lipoma simplex* also occurs. Clinically these two varieties may be

* A paper read before the British Orthopaedic Association, October, 1922.

considered together, because the symptoms, signs, locations, treatment, and prognosis are the same

They are composed of fat tissue without any evident capsule other than the sheath itself to which they are so intimately attached, hence the sheath must be sacrificed in removing the growth. The tumour is liable to surround the tendon within the tendon sheath, and follow it towards its insertion. In these cases there may or may not be fluid within the sheath, and the tendon is intact as a rule. On the other hand, the lipoma may be outside the sheath and attached to it by a pedicle.

They are more frequent on the tendon sheaths of the palm of the hand, but occur also on tendon sheaths in the leg. The commonest symptom is disability, movements with which the tendon is concerned being limited, owing to pain. As with most tendon sheath tumours, diagnosis has seldom been made before operation, the signs being similar to those of tenosynovitis, simple or tuberculous, which is so much more frequent. The diagnosis of ganglion has been made, and lipoma found at operation. The only treatment is removal, and for their ablation it is necessary to remove a portion of the tendon sheath.

ii *Fibromata* are about as uncommon as the lipomata and do not attract any special attention. A specimen of one from a tendon sheath near the ankle exists in the Museum of the Royal College of Surgeons (608). It is the size of a small walnut and lobulated on the surface, and had been growing for twenty years.

iii *Chondromata* are probably less common than the other simple tumours. Meeting with a case of this kind earlier in the year, my attention was drawn to their rarity. Cartilaginous tumours are rare except in connection with bone. They are reported⁴ as occasionally growing in muscles and fasciae. Enchsen refers to three cases the tumours being in the tibialis anticus, vastus externus, and pectoral muscles, and Liston removed a chondroma the size of an orange from the vastus externus.

As tumours of tendon sheaths they are considered worthy of mention by Ombredanne¹ and Delbet.⁵ The latter states that they are quite different from true chondromata of bone. Chauvain and Roux⁶ report a case in detail, the tumour occurring on the tendon sheath of the extensor tendon of the fourth toe. They diagnosed the case as ganglion but found a solid nodule attached to the tendon sheath and had to resect a portion of this sheath to remove it.

Histologically, it was found to consist of hyaline cartilage and fibrocartilage, with bands of fibrous tissue. They consider it to be of the nature of 'traumatic perimusculotendinitis', the history being a sequence of a violent blow followed by ecchymosis, a hæmatoma, and then this tumour.

The history of the tumour in the hand which I report fully is different. The patient was a professional violinist, and she complained of pain and swelling in her left hand for two months, so that she could not play her instrument. She could recollect no definite injury, but this hand she used greatly in playing her violin. There was no doubt about the swelling in the palm. It was in the space between the heads of the third and fourth metacarpal bones, and was soft, non-fluctuant, and did not appear to move with the flexor tendons.

Full flexion of the ring finger was painful, but no limitation of flexion or extension at any of the finger-joints was present. The skiagram showed an area the size of the sesamoid bone at the base of her index finger, situated on the radial side of the neck of the fourth metacarpal bone, which was more opaque than the soft tissues, but less dense than the shadow cast by the bones of the hand. At operation, an encapsulated mass was found on the radial side of the flexor tendons which go to the ring finger. It had a pedicle attached to the flexor tendon sheath, just distal to the metacarpophalangeal joint of the ring finger. The specimen measured $1 \times \frac{1}{2} \times \frac{1}{2}$ inch. It was lobulated, and somewhat grey in colour. The microscopic examination revealed that the tumour was a chondroma. A connective-tissue stroma containing blood-vessels and no abnormal cells surrounded masses of hyaline cartilage. Professor Slattoek was kind enough to examine the section and states that it is a simple chondroma (Fig. 372). The opaque area shown in the skiagram is due to calcification of a portion of the neoplasm.

Calcification occurred also in the following case, which is one of Marcus Beck's. Mr Raymond Johnson⁴ refers to it, and tells me he helped Beck remove the tumour from the flexor tendon sheath of the index finger. It was thought to be a ganglion before operation, and the specimen is now in the Museum of University College Hospital Medical School. Mr E. K. Martin has re-examined the specimen, and owing to his kindness I am able to illustrate a section of the tumour, which was the size of a cherry. It is, like the last, composed of several lobules of cartilage, and there is an area of calcification (Fig. 373).

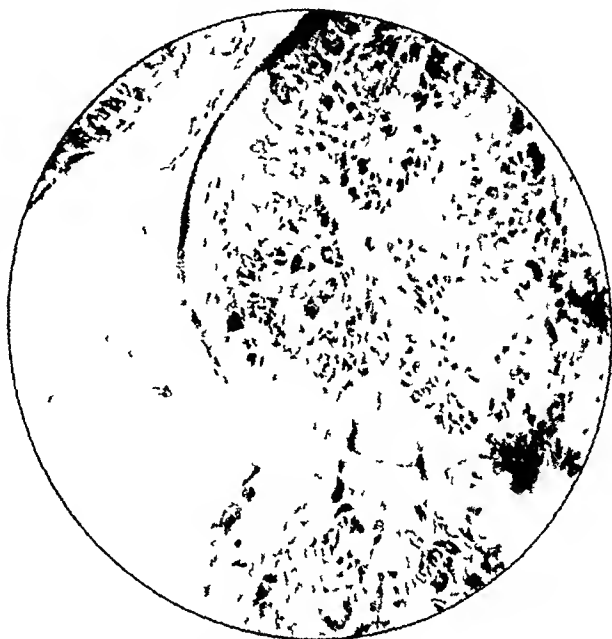


FIG. 372.—Chondroma of tendon sheath ($\times 80$)

A specimen (1569, Old Catalogue) that is in the Museum of King's College Hospital shows several tumours on a tendon sheath. The catalogue states that these are fibromata. Re-examination shows them to be composed of fibrocartilage, and it is to be noted that these tumours are multiple, and less lobulated than the previous two which were examined (Fig. 374).

In regard to the origin of these tumours, one would suppose that cells capable of producing cartilage had strayed from the primitive basis of the bone into the attached connective tissue. Nothing in the microscopic sections suggests that they are other than benign tumours. There is no evidence to show that they recur after removal.

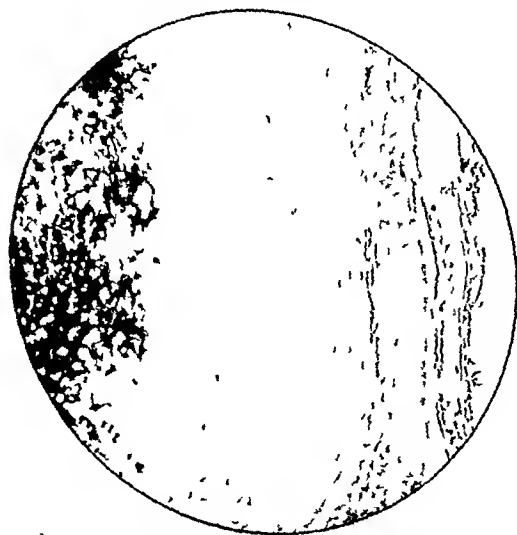


FIG. 373.—Chondroma of tendon sheath showing calcification. This section was prepared and re-examined by Mr E. K. Martin.

(iv) *Ganglion*—This condition should be regarded as a tissue degeneration rather than a cystic tumour. In addition to the favourite site on the dorsal carpal region, ganglion may appear in the palm near the metacarpophalangeal articulations, the site of the greater number of tumours of tendon sheaths. It is interesting to note that a ganglion may form inside a tendon.⁷ This is certainly a point in favour of the pathology of the formation of ganglion worked out by Ledderhose,⁸ and confirmed by Thorn⁹ and Franz,¹⁰ being correct. They state that their origin is in colloid degeneration of hyperplastic peritendinous tissues. Histologically a ganglion is indistinguishable from a lymphangioma, but it is not easy to conceive the idea that this condition is derived from dilated lymphatics.

2 *Giant celled Myeloma*—This is without doubt the commonest tumour of the

tendon sheath, and has attracted considerable attention, as its pathology is of great interest

The common site is over the metacarpophalangeal articulation in the palm, but the forearm tendons are not exempt, any more than are the tendons passing over the ankle-joint. There is usually a history of the part having been injured. The tumours are small, growth is slow, and interference with function is late. They are adherent neither to bone nor to skin. The differential diagnosis is very difficult owing to the lack of special signs or symptoms produced by these and other tumours, by which they could be distinguished from tenosynovitis. Tournieu¹¹ discusses the diagnosis at considerable length in excellent papers, but acknowledges that even should a neoplasm of the sheath be diagnosed, the only evidence to suggest myeloma is small volume, soft consistence, and slow growth. The majority of cases described have not been diagnosed until a section of the tumour has been cut. The growth is lobulated, and has a slight brownish or pinkish coloration—not the grape

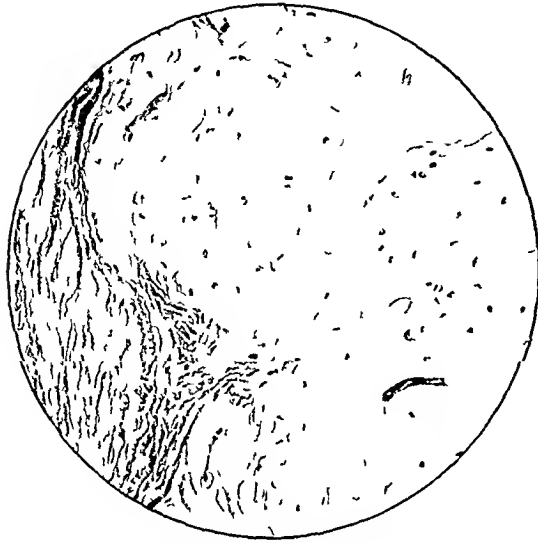


FIG 374—Fibrochondroma of tendon sheath ($\times 80$)

juice colour of the myeloma of bone. Microscopically, it is found to be divided into lobules by strands of fibrous tissue. In the lobules are a variety of cells, including multinucleate giant cells, and pigment.

Broders¹² describes the histology in detail in a recent paper, illustrated with microphotographs. There is evidence that these tumours recur locally, but they must be distinguished from the sarcomata, which in addition to local recurrence, may form metastases. The distinction is only possible in an early case with the aid of the microscope.

The case I have to report is that of a girl who was hit on the back of the right thumb when playing hockey in November, 1921. There was what she calls a 'gash' over the back of the thumb. In December she noticed a small swelling in the same place which gave her no pain, but a dull ache. It has grown slowly. In September, 1922, ten months later, she came for treatment. It was found she had a small swelling, a little larger than a pea, over the radial side of the first phalanx of the right thumb. It was free of the skin—which seemed to be normal—and was movable laterally. It did not appear to be fixed to extensor or flexor tendon when either was brought into action. There was a small nevus over the side of the terminal phalanx of this thumb.

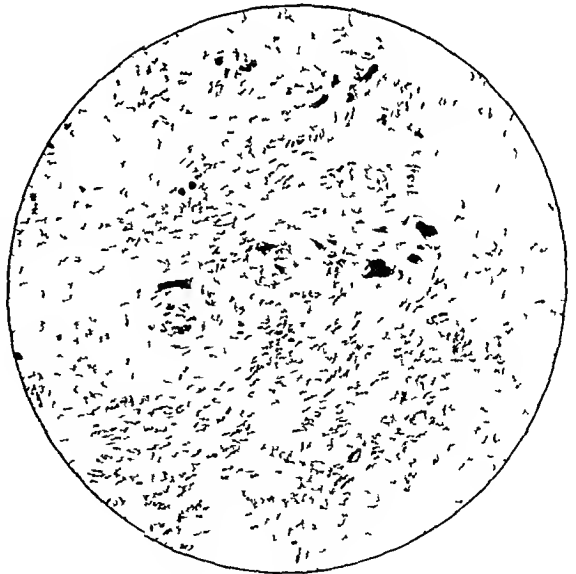


FIG 375—Giant celled myeloma of tendon sheath ($\times 80$)

The tumour was excised under local analgesia. An encapsuled lobulated tumour was removed from the connective tissue deep to the skin. The capsule was attached to the sheath of the long extensor of the thumb and to the tendinous expansion. It felt of solid consistence after removal.

The section shows the tumour to be a giant-celled myeloma, with the typical giant cells in large numbers, as is the case with these tumours (*Fig 375*).

At the Royal College of Surgeons there is one specimen only of a giant-celled myeloma of a tendon sheath (1608-1). It was excised from the flexor aspect of the right thumb, opposite the interphalangeal joint, of a woman, age 24, a clerk. She noticed the swelling when it was the size of a split pea, two years previously.

A portion of the tendon sheath had to be removed in order to free the tumour, which was 1 inch in length and somewhat oval in shape. Its surface is broken up into many lobulations, and is yellowish in the prepared specimen. On section, the deeper portion is white.

The history of the myeloma of tendon sheaths is one of pathological controversy.¹³ The first problem was settled by Heurteux,¹⁴ who separated the myeloma from the sarcoma by the clinical results of the cases. The second problem is as to the exact nature of the myeloma, and this is not yet decided to the satisfaction of all. Professor Shattock tells me that he regards the giant-celled myeloma of tendon sheaths as of the same kind as the giant celled myeloma occurring at the ends of bones. It is suggested that it is due to the embryonic displacement of bone elements into the tendon sheath, which is continuous with the bone. The elements displaced are those related to the marrow. Tournoux supports this theory. The tumours occur in babies as well as in people of 80, and possibly the appearance of the tumours in babies lends support to the theory. Thus being so, the chondroma and myeloma of tendon sheath are comparable in their origin, but neither tumour has a connection with the bone.

Stewart, who has done a considerable amount of work on these tumours, considers that they should be called myeloid endotheliomata, as suggested by Bellamy.¹⁵ He believes the myeloid giant cells to be of tumour origin and an essential part of the growth, but that the minute structure is that of an endothelioma, and that cells of inflammation are not present in the tumour. He points out that too much stress has been laid on the presence of pigment and xanthoma cells, particularly by Dor¹⁶ and Fleissig,¹⁷ whereas these cells occur in other tumours and are dependent on tissue disintegration.¹⁸

The American school, represented by Ely¹⁹ and Broders, find that local extravasation of blood is an almost constant predecessor of the tumour, and regard the growth as a granuloma rather than a neoplasm. The giant cells are accounted for by being regarded as a collection of nuclei surrounded by an irregular mass of hyaline and faintly granular material of foreign-body origin.

3 Sarcoma—This tumour of tendon sheath differs from the giant-celled myeloma in its clinical characteristics, just as the myeloma and sarcoma of bone differ. It is, in fact, a malignant tumour. It starts with local symptoms, the tumour generally being in the line of the tendon, and hard and lobulated on the surface. Sometimes the movements of the joint near the tumour are limited. Pain is not great and is variable. It occurs most commonly, I gather, in the palm of the hand, in the forearm, in the sheath of the peronei muscles, or in the tendo Achillis. Skin attachment is unusual, infiltration into surrounding tissue is the rule. The rate of growth is rapid when the round cell is predominant. Dissemination occurs as in other sarcomata, and the lymphatics are not commonly invaded. If the tumour is removed locally, recurrence follows. Hence amputation of the limb is advised, unless radium or Coley's fluid appeals to the surgeon, as the prognosis is unhappy in these cases. A specimen at the Royal College of Surgeons (No 6071) is that of a chondrosarcoma by the side of the tendo Achillis. This occurred in a girl of 17, and the limb was removed by amputation. The histology shows the growth to be composed mainly of cartilage in which are scattered many irregular foci of calcification. Some of these foci become directly transformed into bone. The matrix of the cartilage is in places fibrous, and bone is likewise developed from such by the

intramembranous method, and the formation of osteoblasts. Here and there groups of branching channels, varying in diameter, have arisen from mucinoid degeneration of the strands of connective tissue, which ramify in the cartilage.

In certain areas the cartilage is replaced by connective-tissue cells in close collections and the tendon itself is infiltrated by similar cells. There is thus no doubt about its nature, but whether it started in the sheath or tendon we cannot say.

CONCLUSIONS

- 1 Primary tumours of tendons are extremely rare, and probably do not occur.
- 2 The giant-celled myeloma is the commonest tumour of the tendon sheath.
- 3 The differential diagnosis between tenosynovitis and tendon-sheath tumours presents great difficulties.
- 4 The giant-celled myeloma—(a) Grows as a rule at the site of an injury, (b) Has the characteristics of a benign tumour, but is liable to recur locally, (c) Is comparable pathologically to the giant-celled myeloma of bone.

The kindness and help afforded me by Professor Shattock and others has been invaluable, and I wish to express my gratitude to them.

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MYOSITIS OSSIFICANS AND VOLKMANN'S PARALYSIS

NOTES ON TWO CASES ILLUSTRATING THE RARER COMPLICATIONS OF SUPRACONDYLAR FRACTURE OF THE HUMERUS*

By W. ROWLEY BRISTOW, London

A CONSIDERATION of the two cases here reported is of interest in illustrating two of the rarer complications of fractures about the elbow.

1 TRAUMATIC MYOSITIS OSSIFICANS

The child, a girl of 6½ years, sustained a supracondylar fracture by a fall in June, 1921. The local doctor reported that he had reduced the deformity under an æsthesia and that the next day massage and mobilization had been commenced (*Fig. 376*).

For a time, all is reported to have gone well, but two weeks after the accident, the elbow became less movable, and the child complained of severe pain on any attempt at movement.

I first saw her three weeks after the accident and, on examination, the region of the elbow was hot, inflamed, and tender. The whole area was hard and indurated, and only a few degrees of movement were permitted, the elbow being practically fixed at a right angle. A lump could be made out in front of the elbow, and x-ray examination (*Fig. 377*) revealed new bone formation, presumably in the brachialis anticus. The arm was put at rest in a 'collar and cuff' support, and all physical treatment discontinued. The 'collar and cuff' consists of two loops—one surrounding the neck and the other the wrist—joined together by a length of bandage, so holding the elbow at any required angle. This contrivance was originally described by Hugh Owen Thomas under the term 'gauge halter'.

After leaving the elbow at rest in this support for a few days, it was possible to bring it gradually into the flexed position by shortening the connecting bandage: no reaction followed the manoeuvre, which took some five or six days to complete.

A skiagram taken on Aug. 30 (*Fig. 378*) shows the shadow of the new bone to be smaller, but more opaque and defined. All inflammatory signs had subsided, and the elbow was allowed to drop by degrees to a right angle, by adjustment of the sling, without causing pain or local reaction, the power of voluntary flexion being well retained.



FIG. 376.—Skiagram taken June 1921.

* A Paper read at the British Orthopaedic Association at the Royal Society of Medicine, on Friday, October 20 1922.

On Nov 16, five months after her accident movement was free, controlled, and painless, from full flexion to a right angle—the degree allowed by the sling. The x ray



FIG 377—Skia gram taken on July 17

showed a great decrease in the new bone (Fig 379). Free movement of the joint was allowed from this date, but no passive or forced movements were permitted.

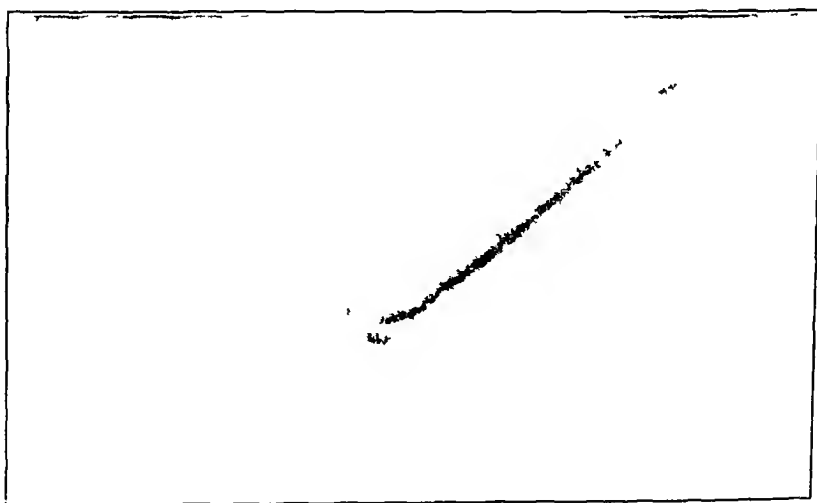


FIG 378—Skia gram taken on Aug 30

When last examined, in April 1922, ten months after the injury the arm was practically normal. Movement through the full range was free and controlled, and the x ray (Fig 380) shows the further decrease in the new bone formation.

This case presents no extraordinary features, but it is of interest as showing the way in which the new bone becomes absorbed and the joint mobility restored when the

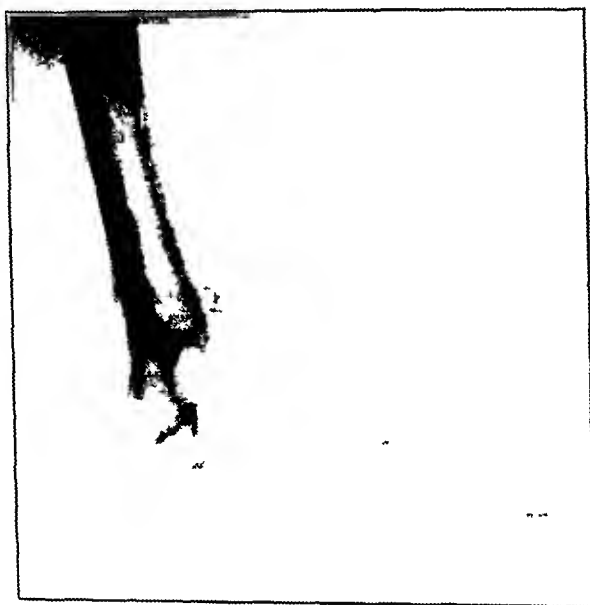


FIG 379 —Skiagram taken on Nov 16

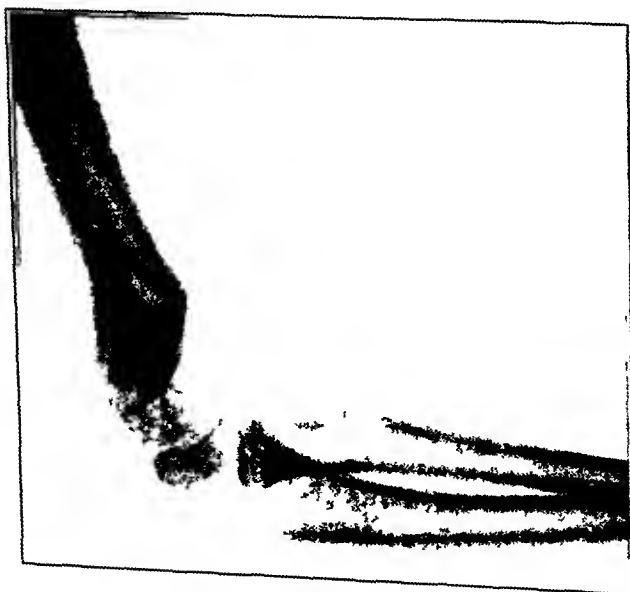


FIG 380 —Final skiagram, April 2 1922

parts are put at rest. A consideration of the series of skiagrams may be of service when we are called upon to give a prognosis in a similar case.

2 VOLKMANN'S CONTRACTURE

The second complication is Volkmann's contracture. This calamity is fortunately rare, and in many cases preventable, but it is not so in all. As is well known, it is usually associated with the pressure of tight bandages and splints. Cases are reported, however, in which the condition has ensued on accidents when no splints or bandages have been applied. A full account of the condition and its etiology, together with references to the literature up to that date, is given by Dudgeon². More recently an experimental study of the subject has been made by B. Brooks,³ who summarizes his conclusions as follows: "That the classic picture of Volkmann's ischæmic paralysis could only be explained on the basis of acute venous obstruction would seem quite clear."

Suffice it to say here that the condition is reported to have followed the use of an Esmarch bandage, rupture or contusion of the main vessel of the limb, and thrombosis of the axillary artery.

Volkmann, in his article published in 1875, considers that the paralysis and contracture are caused by a cutting off of the blood-supply, and that the muscle dies.

The microscopical examination of portions of forearm muscle from the case about to be reported, as well as from similar cases, upholds this contention. The microscopic drawings figured illustrate this point particularly well when compared with those taken from a case of degeneration following division of a motor nerve. The condition is essentially different from that of degenerated muscle which has resulted from interference with the lower motor neurone.

In Volkmann's contracture there need be no interference with the peripheral nerves, and in consequence, no sensory change. In point of fact the nerves are not infrequently involved, and it is therefore not uncommon to find an area of sensory loss.

In May, 1922, the patient, a boy, age 8, was referred to me. He had sustained a supracondylar fracture of the humerus three months earlier. The accident had happened in January and the deformity was said to have been reduced under chloroform anaesthesia two hours later. The arm was bandaged with the elbow flexed. The arm swelled the same night and the fingers were blue. The bandages were cut down after twenty-four hours when the arm was blue and very swollen, blisters appeared next day, and the hand became contracted. Somewhat vigorous treatment had been applied, and I examined the boy for the first time three months after the injury.

He presented the appearance of a typical Volkmann's contracture (*Fig. 381*). The wrist-joint was flexed, the metacarpophalangeal joints were hyperextended and the interphalangeal joints flexed. The deformity was fixed. The forearm muscles were hard, much wasted, and brawny, the elbow was practically fixed at a right angle, and a deep scar, the site of the original blister, crossed the external border of the forearm in the middle and upper thirds, and was down to, but not adherent to, bone. Anaesthesia was complete in the median and radial areas, incomplete in the ulnar. On electrical



FIG. 381.—From photograph showing the Volkmann's contracture.

examination the ulnar muscles were normal in reaction, but no faradic or galvanic response could be obtained from the median muscles

The lesion was therefore diagnosed as Volkmann's contracture with complete physiological division of the median and radial nerves. The radial anaesthesia was obviously explained by the scar crossing the radial nerve, which was causing the interruption

X-ray examination showed a supracondylar fracture of the humerus, with the usual gross displacement. The lower fragment was drawn upwards and displaced backwards, and the sharp lower end of the upper fragment projected into the antecubital fossa (*Fig 382*)

It was decided, after a neurological consultation, that the median nerve should be explored. At the operation the nerve was found to be completely divided, with the usual end-bulb formation, and the division was obviously caused by the sharp projecting end of the upper fragment. It seems

probable that this lesion, which is uncommon, was caused, not at the time of the accident, but rather at the subsequent manipulation. The forearm muscles were hard and yellow, and did not look like muscle at all. They formed an almost complete plaque of tissue, and it was not possible to separate the individual muscles. Suture of the nerve was effected without tension, and was worth while in view of the probable recovery of sensation in the hand. The lesion having occurred in the position in which the branches to the forearm muscles are given off precludes the possibility of their recovery, even if the state of the muscles had allowed of this.

The further treatment of the patient was directed to a reduction of the deformity by splintage. The fingers could be extended when the wrist

was fully flexed, and were held extended by a small splint made of plaster-of-Paris moulded to the hand, extending from the tips of the fingers to the wrist. A metal splint was then applied, taking purchase from the flexor aspect of the forearm above, and from the hand splint below. A felt pad was placed over the dorsum of the flexed wrist, and this was firmly bandaged to the metal splint. The result of the action of this force is gradually to extend the wrist, the fingers meanwhile being held extended by the hand splint. The splintage was maintained day and night for some three weeks, when the deformity was corrected. Examination some months later revealed signs of commencing sensory recovery in the median nerve.

At the operation, portions of muscle, or what had been muscle, were removed. Dr Greenfield cut sections of this, and we have had drawings prepared from typical sections, and at the same time, drawings of a transverse section of normal muscle and of muscle degeneration following division of a peripheral nerve (*Figs 383-386*)

The relative rapidity of the changes—the death of muscle in the ischaemic case taking place at the time—is manifest by a consideration of the sections, for, although there was a complete division of the median, the changes shown are not those of muscle degeneration due to peripheral nerve division, but of death of muscle and replacement by fibrous tissue

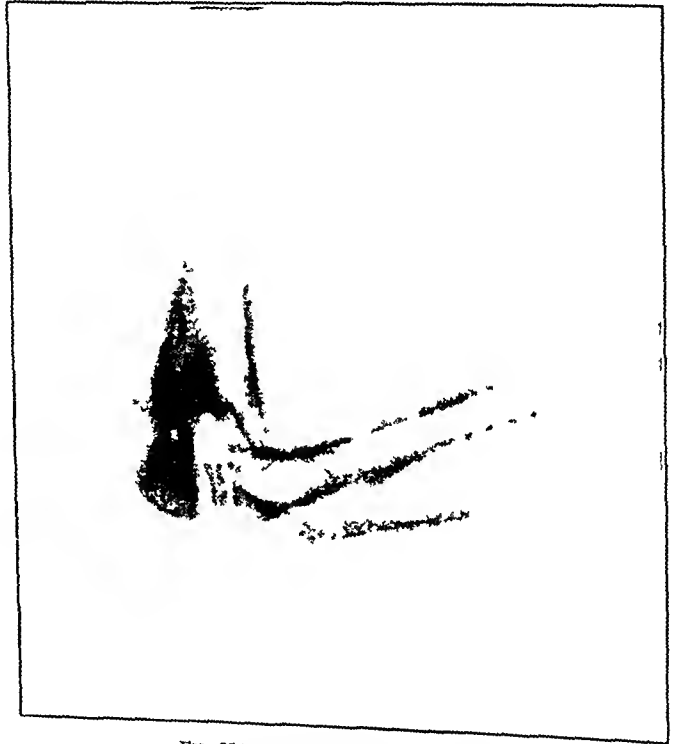


FIG 382—Skilogram illustrating Fig 381

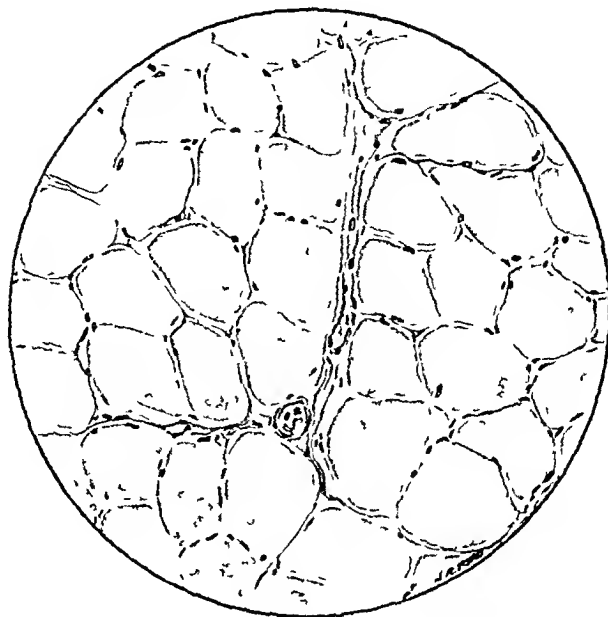


FIG. 333.—*Normal Muscle*. Transverse section showing (a) the muscle bundles and their arrangement, (b) the sarcolemma nuclei, (c) the scanty fibrous tissue-stroma.

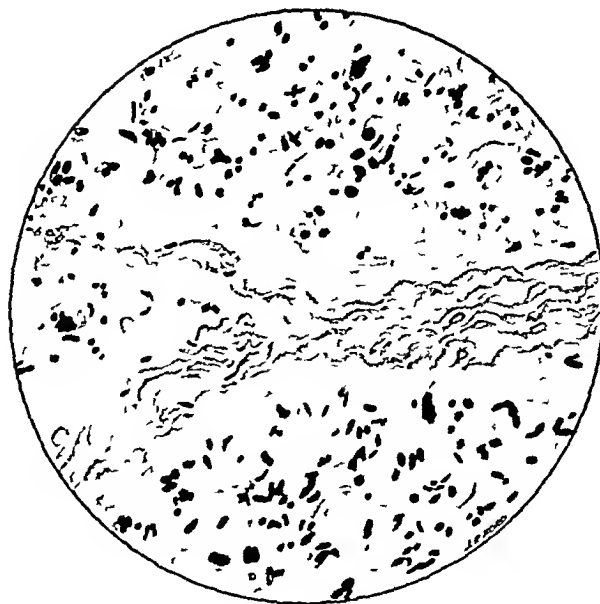


FIG. 334.—*Degeneration of Muscle* (after division of a peripheral nerve). Transverse section showing (a) the muscle fibres shrunken, (b) the nuclei of the sarcolemma multiplied and in some cases passing into the fibre, (c) the thickening of connective-tissue septa. (In longitudinal section this muscle shows no cross-striation. The muscle is capable of recovery.)



FIG 385.—*Transverse Section of Muscle from the Patient, showing (a) many oval fibres which do not fit in so well together as normally (b) no sarcolemmal nuclei, (c) a slight thickening of fibrous tissue and absorption by cells of connective tissue working in from the edge, (d) the very slight, if any, diminution in width of fibre (The cells from the connective tissue have absorbed the muscle fibre and laid down connective tissue in its place. There is some muscle detritus left at the edge.)*

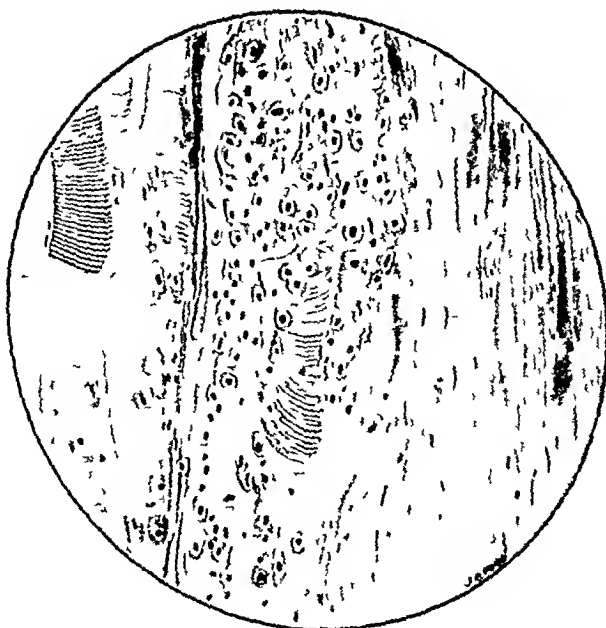


FIG 386.—*Longitudinal Section of Muscle from the Patient. Passing from left to right points are noticed (a) dead muscle bundles in which cross-striation is very in some sections at the cross-striation (b) a few nuclei of cells spreading along the muscle up into fragments, and absorbing the remains, (c) the fibrous tissue is laid down in regular bundles, replacing nuclei bundles which have been removed by the phagocytic cells. (d) the fibre is the fibre breaking bundle,*

REPORT ON A PIECE OF MUSCLE FROM THE FLEXORS OF THE FOREARM

The muscle was received fresh within an hour of the operation. One piece of it was fixed in formalin, another in trichloroacetic mercuric chloride fixative (Heidenhain), and another in Zenker's fluid. The pieces fixed in formalin were treated by the Weigert Pal method for myelinated nerve fibres, but none of these could be seen in any sections examined. The pieces of muscle fixed by the other methods were embedded in celloidin, and longitudinal and transverse sections were cut and stained by hæmatoxylin with van Gieson's counterstain. All showed a similar condition.

The most striking histological feature about the muscle was that over large areas no nuclei of any sort could be seen. This applied not only to the sarcolemmal nuclei, which seemed to have disappeared completely everywhere, but also to the nuclei in the interstitial fibrous tissue, muscle spindles, and blood vessels. At first sight one was inclined to blame the hæmatoxylin, as the differential staining of the picrofuchsin for muscle and fibrous tissue was well preserved. But where the sheath of the muscle or its tendinous attachment was included in the section, the nuclei in these structures stained perfectly. One had to conclude, therefore, that the nuclei had in reality completely disappeared from the muscle bundles and the other tissues contained in them. The muscle fibres, although they retained their normal arrangement, were shrunken and more rounded than normal. The cross striation was extraordinarily coarse, and in some sections the fibres appeared to be fissuring across at the lines of the cross striation.

The fibrous tissue between the muscle bundles was slightly thickened, but scarcely enough to constitute an abnormality. At the edges of the muscle where it was surrounded by its sheath, and at its junction with its tendon, there was everywhere a collection of round cells which lay between the muscular and fibrous tissue. These cells formed an almost continuous ring, usually not more than one or two cells deep, around the outside of the muscle. In some places they could be seen penetrating between the muscle fibres for a short distance from its outer border, but they never went deeper than about the third layer of muscle fibres.

Viewed under a higher magnification these round cells could be seen to be invading the muscle fibres. All stages of this process could be seen. The cells at first were lying closely applied to the muscle fibres. Then they became more rounded and passed gradually into the muscle fibre, which at the point of invasion lost its transverse striation and became granular. More cells then invaded the fibre, until over a considerable extent it was converted into a granular mass containing many cells, and only showing here and there the remains of its transverse striation. Then fibroblasts appeared, and fibrous tissue was laid down, at first in very thin threads and later more densely (*see Fig. 336*).

These processes led to a thickening of the fibrous tissue of the sheath, which, near the edge of the muscle, had a remarkable resemblance to muscular tissue. This appearance was best seen in transverse sections, which showed an arrangement of the connective tissue fibres in rounded strands, often containing one or more nuclei, and in some cases showing inclusions of brownish granular material. The latter seemed to be the remains of muscle fibres which were undergoing absorption, and the appearance suggested that the fibrous tissue cells were laying down fibroblastic fibres in the position of the muscle fibres which had been absorbed (*see Fig. 385*).

The whole process seemed to be one of absorption and replacement of dead muscular tissue by fibrous tissue—the muscle acting as a non-septic, non-irritating foreign body, and being treated as such by the surrounding tissues.

It was impossible from the material at our disposal to say how far this process had gone, but apparently a considerable increase in the thickness of the sheath of the muscle had taken place. The process must necessarily be a slow one, as it was working only from outside the muscle and only to a very slight degree from the connective tissue septa within the muscle. The latter, indeed, seemed to have died along with the muscle fibres, as they also contained no nuclei.

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The prognosis would seem to depend mainly upon the severity and extent of the original lesion, and in severe cases must always be bad. The deformity can be overcome by appropriate measures, but a return of voluntary power is not to be expected. Any power that is present will, under treatment, be conserved and will improve, but total inability voluntarily to flex the fingers to even the smallest degree, weeks or months after the onset of the contracture, is a very grave prognostic sign. The most that can be hoped for in such a case is to correct the deformity, which should always be possible, and possibly to restore sensation in an anæsthetic area.

In conclusion I would express my indebtedness to Dr Greenfield who prepared and cut the sections, and who has written a note on the pathological findings.

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LENGTHENING OF THE TENDO ACHILLIS

By H. H. GREENWOOD, SWINDON

THE reliefment of this operation by simple transverse section of the tendon is gradually falling into deserved disfavour. The gap caused by the wide separation of the severed ends of the tendon can only be bridged by fibrous tissue, with the not-surprising result that there is left undue weakening of this powerful tendon, in many cases necessitating an operation for repair of the defect.

Hemisection of the tendon at two points distant from each other one inch or more was a definite improvement, but still the operation was done subcutaneously by a tenotome. It was believed that the resulting fracture of the tendon could be represented by *Fig. 387*.

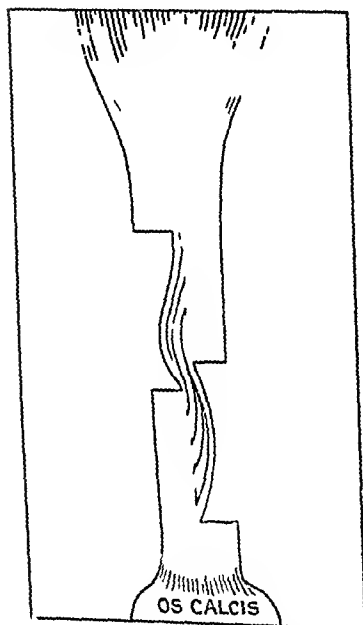


FIG. 387.—Hemisection of tendon ideal result.

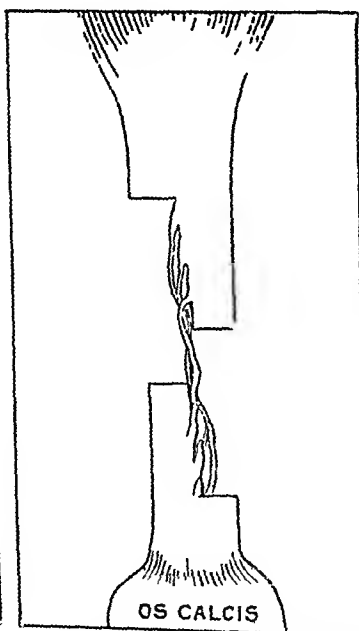


FIG. 388.—Hemisection of tendon showing the snapping of one limb that frequently results.

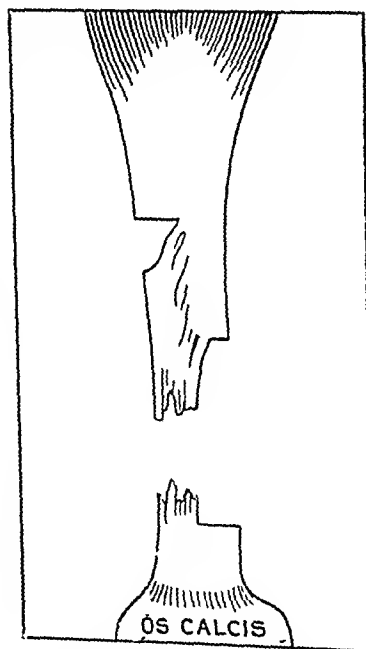


FIG. 389.—Hemisection of tendon showing resultant wide separation of ends.

In reality the usual result is either snapping of one of the limbs of the incised tendon, as in *Fig. 388* or wide separation of the ends, as in *Fig. 389*. Indeed, if the points of hemisection be more than one inch apart, the force required to produce a sliding tear is considerable, so much so that it is no matter for surprise that the condition shown in *Fig. 388* is left. If we consider the mathematics of the question we shall see that much wider separation of the ends must occur than is imagined by those who still adhere to the closed operation. The undesirability of leaving a wide gap is realized by Calot,¹ for he says, "One ought to divide when it is merely a question of obtaining a lengthening of 1½ cm in a child or of 2½ cm in an adult, because Nature may (not will) replace this amount of separation. But if you ought to obtain more than that you will perform elongation of the tendon."

The reality of this wide separation between the divided ends is not merely idle speculation, for repeatedly, after doing the usual hemisection by a closed tenotomy, and rotating the foot into the desired position the marked depression that appeared on the site of section has impelled me to proceed to make a longitudinal incision and so put the matter to the proof. Invariably after exposing the divided ends I have found the condition depicted in *Fig 388* or *Fig 389*.

Other surgeons have been driven by like considerations to adopt the open operation, deliberately lengthening the tendon to the extent demanded by the condition of the foot and only exceptionally to do a closed tenotomy.

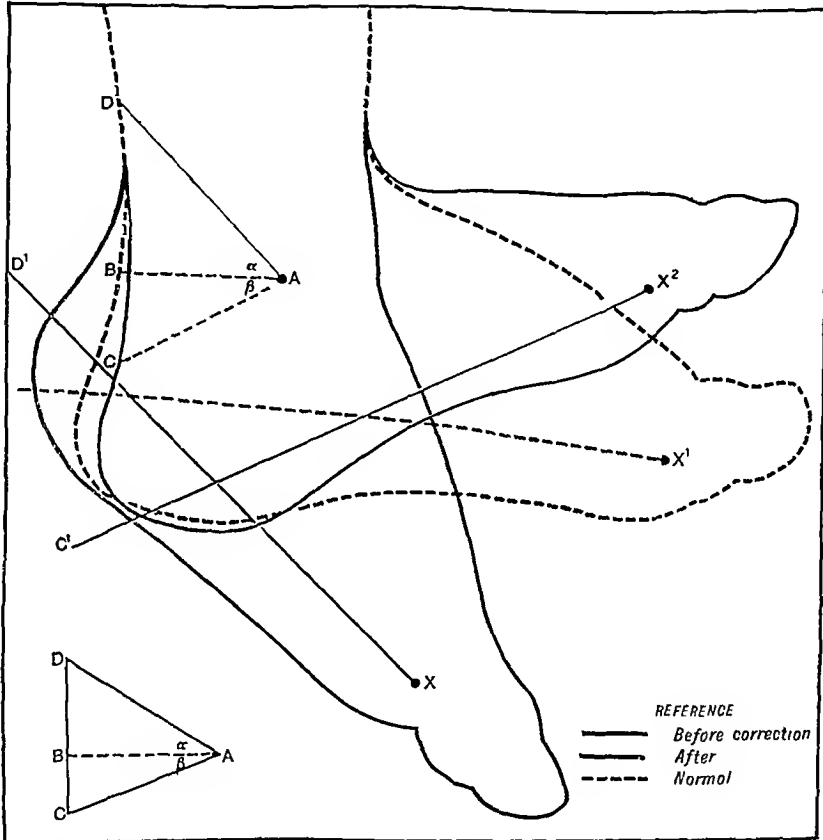


FIG 390.—Diagram of foot illustrating method of estimating the required amount of lengthening of the tendon.

Mr Fairbank² in an article on the 'Orthopædic Treatment of Polomyelitis,' writes "I prefer the open method—it is more accurate, and the ends of the tendon can be sutured so as just to allow the foot

No further justification for the open operation seems necessary, but there remains the question, hitherto unanswered, "By what means is it possible to estimate beforehand the extent to which the tendon in any given case requires lengthening?" An adequate reply will certainly lead to greater precision and this I have attempted to furnish.

The ankle-joint allows a hinge-like movement round a horizontal coronal axis, which passes through the centre of the internal malleolus, this point lies further forward than is commonly depicted or appreciated.

In an investigation carried out by taking tracings of the feet of 60 normal children

at ages from 1 to 12 years, the average distance of the axis from the posterior border of the tendo Achillis (AB in *Fig 390*) was found to be —

From 0 to 2 years (10 examples)	$1\frac{1}{4}$ in
2, 4, ,	$1\frac{1}{8}$ "
4, 6, ,	$1\frac{1}{2}$ "
6, 8, ,	$1\frac{1}{2}$ "
8, 10, ,	$1\frac{1}{2}$ "
10, 12, ,	$2\frac{1}{4}$ "
In 10 adults (5 male, 5 female)	$2\frac{1}{4}$ "

A foot in a condition of talipes equinus lies at an angle which departs from the normal right angle to a variable extent—a very usual one is 45° . To obtain the best result, the foot should be moved round the axis of the ankle-joint until it passes the right angle to the extent of some 30° , it is thereby made to traverse the full range of movement possible to a normal foot. It will be necessary therefore to move the deformed foot through an angle of 75° altogether.

In carrying out this movement, a given point D (*Fig 390*) on the tendo Achillis will move downwards along the arc of a circle whose centre is the axis of the ankle-joint and whose radius is AD, to a point C. For practical purposes we may take the arc of this small segment as a straight line DBC. The line AD, meeting the posterior margin of the tendo Achillis at D, is drawn parallel to the longitudinal axis of the foot (which passes through the metatarsophalangeal joint X). The angles α and β are known, AD, AB, AC are approximately equal, AD can be measured, AB can be taken from the average in the above table, and the distance DC (the extent to which we desire to lengthen the tendon) can be computed with sufficient accuracy for our purpose.

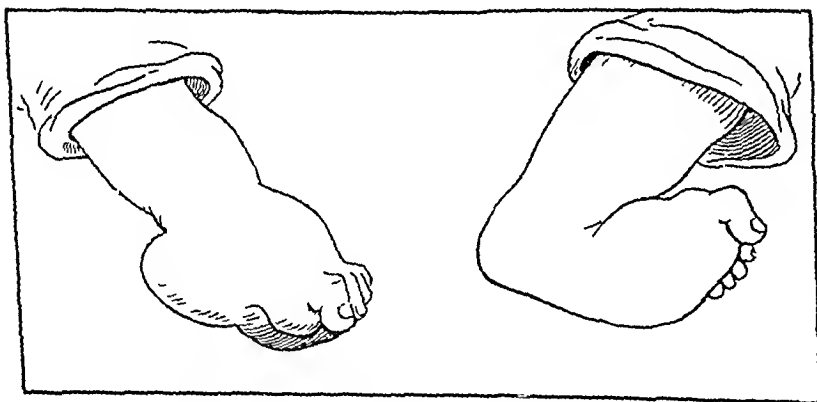


FIG 91.—Talipes equinus in a child of 1½ years before and after operation

For the sake of argument suppose the angle $BAD = 45^\circ$, and $BAC = 30^\circ$. We know that the average measurement of the line AB in a child of 5 years is $1\frac{1}{2}$ in, then

$DC = DB + BC$. But $DB = AB \tan \alpha$, and $BC = AB \tan \beta$. Then

$DC = AB \tan \alpha + AB \tan \beta$. But $\tan \alpha (45^\circ) = 1.0$

And $\tan \beta (30^\circ) = 0.5774$

That is, $DC = 1\frac{1}{2} \text{ in} \times 1 + 1\frac{1}{2} \text{ in} \times 0.5774 = (\text{roughly}) 1\frac{1}{2} \text{ in} + \frac{3}{4} \text{ in} = 2\frac{1}{4} \text{ in}$

It will be seen, then, that in a child of 5 years a gap of $2\frac{1}{4}$ in must be allowed for, and if it be desired that the ends should slightly overlap before suture, quite $2\frac{1}{2}$ in. For an adult in whom $AB = 2\frac{1}{2}$ in the gap will be about $3\frac{3}{8}$ in.

If it be objected that 45° for the angle DAB is extreme and we take an angle of 30° , then for a child of 5 years the gap will need to be $1\frac{1}{2}$ in.

That these figures are substantially accurate I have confirmed repeatedly by actual measurements during the course of an open operation.

The angles chosen as examples represent those of a case of moderately severe talipes equinus but the angle α sometimes approaches 60° . Reference to *Fig 391*, which is a

drawing from a not uncommon type of case in a child of $1\frac{1}{2}$ years, will support my contention that the angles assumed for the sake of argument are not excessive. The positions shown are before and after operation.

In the table subjoined I have worked out approximately the length of the gap that will be left in the case of three common angles at which the foot is found. The angle α varies but the angle β remains constant. Given the premisses, it will be easy to compute these distances with greater accuracy if it is found desirable.

TABLE OF APPROXIMATE GAPS BASED ON AVERAGE MEASUREMENTS

AGE	AVERAGE MEASUREMENT OF AB	LENGTH OF GAP		
		Angle DAB = 40°	Angle DAB = 30°	Angle DAB = 10°
0 to 2 years	$1\frac{1}{2}$ in	2 in	$1\frac{1}{2}$ in	1 in
2 4	$1\frac{7}{16}$	$2\frac{1}{16}$	$1\frac{7}{16}$	$1\frac{1}{16}$ "
4 6	$1\frac{1}{2}$	$2\frac{1}{2}$ "	$1\frac{1}{2}$	$1\frac{1}{2}$ "
6 8	$1\frac{3}{4}$	$2\frac{3}{4}$	$1\frac{3}{4}$ "	$1\frac{1}{4}$ "
8 10	$1\frac{1}{2}$	$2\frac{1}{2}$	$1\frac{1}{2}$	$1\frac{1}{2}$
10 " 12 "	2	3	2	1\frac{1}{2} "
Adult	$2\frac{1}{2}$ "	$3\frac{1}{2}$	$2\frac{1}{2}$	$1\frac{11}{16}$

Tan $15^\circ = 0.2679$
Tan $30^\circ = 0.5774$

Tan $45^\circ = 1.00$
Tan $60^\circ = 1.7321$

Tan $75^\circ = 3.7321$
Tan $90^\circ = \infty$

It is difficult to appraise with exactitude the results obtained by the older methods, since they certainly do allow correction of the deformity to be achieved. Their chief defect lies in an unnecessary weakening of a limb that is already, as a rule, lacking in power. An open operation deliberately planned on the lines advocated above provides the enfeebled calf muscles with a tendon that is as nearly strong as the untouched one as it is possible to make it.

In doing the open operation, too, it cannot escape notice that there is usually brisk hemorrhage, easily arrested and therefore of small moment, but in a closed tenotomy a considerable effusion of blood must often occur, hidden, but fated to organize and to leave unnecessary stiffness of the joint.

My own improved results, since adopting the open method for all but the mildest cases, leave me in no doubt as to the wisdom of this course. I will only add that a slightly curved incision, lying to the outer side of the tendon, gives the best exposure and causes the least hemorrhage. A light plaster of-Paris case applied immediately over a bandage made by cutting strips from common draper's wool is desirable. A continuous 'blanket' suture of 0 catgut gives good approximation of the skin edges, and can be left untouched until the plaster is removed.

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FAIRBANK *Brit. Med. Jour.* 1921 April 9 517

OSTEITIS FIBROSA.

By R. LAWFORD KNAGGS, LONDON

(Being the Hunterian Lecture delivered at the Royal College of Surgeons on January 29, 1923)

OSTEITIS FIBROSA is the name given to a disease of bone in which part of the osseous framework and its contained marrow are replaced by fibrous tissue. In this, ossification usually proceeds in a more or less scattered manner—in some cases to such an extent as almost to reconstitute the bone.

As the microscopic appearances have some resemblance to those seen in other conditions, it materially assists the diagnosis if the lesion is so gross that the fibrous state and texture can be recognized by the naked eye. The conception of the nature of the affection is somewhat hazy, and cases are to be found recorded under various titles.

Though its name implies 'inflammation', yet its inflammatory character is accepted dubiously and with reservation. A short description of certain allied conditions of whose inflammatory origin there is no question will, therefore, be a useful introduction to the main subject.

OSTEITIS FIBROSA ARISING BY EXTENSION FROM A JOINT AFFECTION, OR AS A RESULT OF SEPTIC IRRITATION

In 1883 Arbuthnot Lane exhibited, at a meeting of the Pathological Society, the heads of both the femurs of a man, age 50, who had suffered from rheumatic arthritis of the hips. In the vertical section a mass of fibrous tissue was seen to extend inwards from the ligamentum teres and to blend in places with the under surface of the articular cartilage, which showed fibrillation at the point of contact. The changes were symmetrical.¹ Similar fibrous patches in the vicinity of rheumatic arthritic joints are described by Ziegler,² and the same author has depicted a cyst in a fibrous area surrounded by bony trabeculae, in the same disease.³ Again, Strangeways has pointed out that skiagrams of rheumatic arthritic joints will sometimes reveal transparent areas in the bones entering into their formation. These are found to be erosions of bone, or cavities in its interior filled with a gelatinous mucoid substance.⁴

The way in which such cysts originate has been described by Nicholson. When they form in the articular cartilage it is by liquefaction of the matrix, the disappearance of the corpuscles, and the formation of a fibrocartilaginous wall. When they occur in bone a corresponding degenerative change takes place. The bone trabeculae in a limited area stain very slightly with eosin, and all traces of bone corpuscles and lamination are lost. These trabeculae undergo resorption by large numbers of giant cells. The bone surrounding such necrotic areas is healthy, and the marrow fibrous, contrasting with the fat-marrow elsewhere. This fibrous marrow zone contains many leucocytes, but no giant cells. We may therefore presume that the mucoid contents of such a space are the result of mucoid degeneration of the intertrabecular tissue and the vanishing bone.⁵

Analogous fibrotic changes may also originate from septic irritation.

In St Bartholomew's Hospital Museum is a superior maxilla whose alveolar process, greatly increased in size, is transformed into a dense bony mass which reaches to the floor of the antrum, though that cavity has not been affected. Small scattered patches of fibrous tissue can be seen on the face of the section through the altered bone. The specimen (400b) was removed from a woman, age 38, who had noticed the swelling for twelve months. Some decayed teeth had been extracted three weeks before, and the

largest fibrous patch surrounds the alveolus which one of them had occupied. The microscopic structure was that of dense cancellous bone with its spaces filled by fibrous tissues showing some signs of mucinoid degeneration.

Thus we see that a fibrous osteitis may be an adjacent complication of different forms of inflammation, and that in some cases localized areas of degeneration may end in cystic spaces with fibrous boundaries instead of patches of fibrous tissue.

OSTEITIS FIBROSA AS A PRIMARY AFFECTION

Whilst it is clear that fibrous osteitis of a secondary character may be definitely associated with inflammatory lesions, it must be admitted that the precise nature of the changes in *primary osteitis fibrosa* is not so evident. The latter variety is a distinct clinical entity, is very liable to be confounded with osteomalacia, osteitis deformans, or with central bone tumours, and is very puzzling to the practitioner.

Its recognition is due to von Recklinghausen, who described and illustrated cases of it,⁶ and Bloodgood⁷ and Elmslie⁸ have added to our knowledge. Bloodgood dealt primarily with bone cysts, but he incidentally defined various forms of osteitis fibrosa. His classification is instructive, and as I shall have occasion to refer to it subsequently its principal groups may be quoted. He describes—

1 Single cysts in which there is no connective tissue lining the cavity. It is always possible to find in such cysts a new connective tissue between the bone lamellæ of the shell (osteitis fibrosa).

2 Cysts with definite connective-tissue lining, which can be peeled off from the bony shell, and is identical microscopically with the fibrous tissue in the bone shell of the first group.

3 A small cyst or cysts in a solid mass of fibroid tissue. The medullary cavity is filled with the same kind of fibrous tissue as that in groups 1 and 2.

4 No cysts, but the bone shell filled with a solid mass of fibrous tissue.

5 Multilocular cysts. The distended shell of the bone is partitioned into multiple cavities which contain either fluid or fibrous tissue.

In this paper however the disease has to be studied as a whole, and in proper perspective. The clinical cases, many of them classical ones,* which will be utilized for this purpose, fall naturally into one or other of the following groups—

- I Those in which the lesion is represented by a uniform mass of fibrous tissue.
- II Those in which a solid mass is showing signs of degenerating into one or more cysts.
- III Cases in which much bone is developed and the disease shows some signs of coming to an end.
- IV Single cysts of bone.

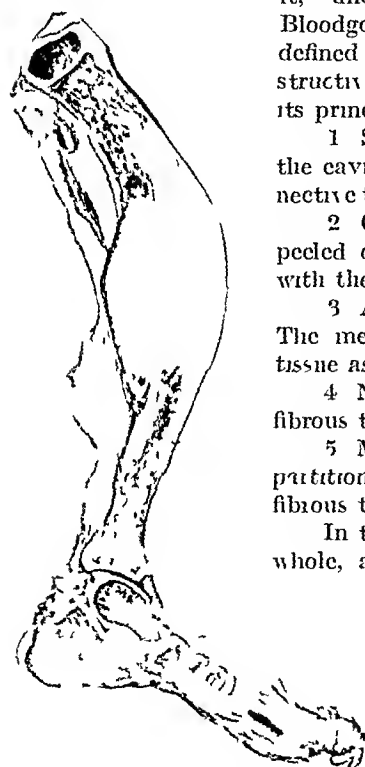


FIG. 392. Tibia and fibula (Bilton Pollard's Case, University College Hospital Museum). (Reproduced from the *Brit. Jour. Surg.* 1911, a, 29.)

Group I—CASES IN WHICH THE DISEASE IS REPRESENTED BY A UNIFORM MASS OF FIBROUS TISSUE.

Case 1—The specimen in Bilton Pollard's case was removed from a child, age 5, who injured her leg when she was a year old (*Fig. 392*). A year and a half later the bone was noticed to be swelling, but was not painful.

* Several of these cases were recorded under other titles before osteitis fibrosa was fully established as a definite disease.

A solid mass of fibrous-looking material occupies the middle third of a bisected tibia, and sends extensions towards both cancellous ends. The bone is much enlarged in its middle portion, and bent like a bow. A small enlargement presenting similar characters exists in the fibula.

Sections showed anastomosing bone trabeculae enclosing spaces filled with a material resembling the fibrillar matrix of growing bone (Sp 1341D, R C S Museum)^{9 10}

Case 2—Bloodgood gives a beautiful photograph of a solid mass of fibrous tissue which Kammerer, of New York, curetted from the femur of a man, age 20. The skiagram showed it to have occupied the shaft in the vicinity of an old healed fracture.¹¹

Case 3—Elmslie records the case of a girl, age 18, in whom he curetted a cyst in the neck of a bent and shortened femur, and three weeks later removed a mass of firm fibrous tissue from the shaft.¹²

When the disease has reached this stage, the bone in section shows a solid area of fibrous tissue sharply differentiated apparently from healthy bone. But an earlier stage no doubt exists when the original bone structure is in process of disappearance. Of this stage we have but little knowledge, because, owing to the insidious and chronic character of the disease, the condition is not recognized until it has produced some deformity or led to fracture.

Group II—CASES IN WHICH A SOLID FIBROUS MASS SHOWS A TENDENCY TO DEGENERATE AND FORM CYSTS

Case 4—The most remarkable example of the association of cysts with osteitis fibrosa is the specimen presented to the College of Surgeons by W T Clegg, and investigated by Eyc (Fig 393). Originally thought to be a sarcoma, it is now catalogued as a soft fibroma, but when osteitis fibrosa began to excite attention, Sir Frederick Eyc, I believe, accepted that diagnosis. It was removed from a man age 24 who ten years before had sustained a fracture of the tibia near its middle. A lump remained at the seat of fracture, and the leg always ached. Nine months before operation the leg began to swell and give pain, but the man walked until admission.

The preparations (R C S Museum 1968, 1 and 2 Gen Path Series) are the two halves of an enormously expanded tibia—the one a macerated and the other a wet specimen.

The former is illustrated in Bland Sutton's *Tumours* (Fig 54, p 94, 6th edit). The latter shows the expanded tibia filled from end to end with a fibrocystic mass. The whole of its upper half is converted into four or five large cysts; the lower half, with some cysts, is for the most part solid and of somewhat homogeneous appearance though in many places a fibrous structure is apparent. All that is left of the bone is a reticulated shell and a small area of cancellous tissue beneath each articular cartilage. There is no sign of a capsule, and the edge of the solid tissue is at places definitely irregular, whilst delicate streamers from the mass can be recognized penetrating the bony shell at the side.

Under the microscope the solid part is seen to be composed of well-developed fibrous



FIG 393.—Fibroma. To illustrate the association of cysts with osteitis fibrosa. (Spec 1968 R C S Museum, Gen Pathol Section)

tissue which in places is showing signs of degeneration. The cysts contained yellowish turbid fluid rich in cholesterol.¹³

Case 5—A femur showing a small cyst developed in a large fibrous inset is pictured by von Recklinghausen in his monograph¹⁴ (Fig 394). The case from which it was taken had other features that make it worth while to record it, and I am indebted to Elmslie's paper for the following abstract—

"The skeleton of a woman, age 66, who died of pneumonia. There was general hyperostosis of the skeleton, with cyst formation, enormous hyperostosis of the skull, hyperostosis and bending of the femora and the right humerus, and porosity of other bones e.g., of the ribs. The bending had not arisen from previous fractures. In the bone marrow there were patches of bone of ivory hardness, patches of spongy bone, islands of fibrocartilage, marrow tissue and large cysts."

Case 6—A femur very similar to the above is shown by Kuster from a female, age 17, and in addition to small cysts there is a circumscribed lobulated mass of cartilage occupying the neck. The last point is of some interest in connection with cyst formation in these cases.¹⁵

It would seem to be the usual thing for ossification to be going on in the fibrous areas except in those cases or in those areas in which there is evidence of early and rapid degeneration (*Case 4*). It is very variable in its incidence, the ossific points being numerous in some microscopic sections, and very sparse or absent in others.

Occasionally definite masses of new bone, sometimes dense and sclerosed, and evidently marking older patches of the disease, can be seen, but as a rule the production of ossific material in *Groups I and II* is not sufficient to produce a striking change in the naked-eye appearance of the fibrous material, though it is often sufficient to cause a feeling of grittiness when the flat side of a knife is drawn over it.

A later stage of the disease is to be recognized in the cases in the next group.

Group III—CASES IN WHICH MUCH BONE IS DEVELOPED AND THE DISEASE SHOWS SIGNS OF COMING TO AN END

In two of the three cases that follow the disease had been in progress for many years, and in the third it developed in adult life—a very unusual thing as the onset is nearly always in childhood, or at least whilst growth is still going on.

Case 7—This was recorded by Shattock and Bernard Pitts in the *Transactions of the Pathological Society*.¹⁶

A woman, age 37, after an injury when she was 31, developed a painful tibia which eventually led to amputation. The tibia in its upper half was transformed into a minutely cancellous bone-like tissue so devoid of lime salts that the bone was as pliable as india-rubber, and was cut readily with a knife. The compact wall and medulla were replaced by this tissue, and the medullary cavity was filled. The lower limit was abrupt, and the bone below normal. Under the microscope the soft tissue in the bone proved to be a highly cellular connective tissue, and the bone trabeculae osteoid in character, their central portions being surrounded with a zone of calcified matrix. (St Thomas's Hospital Museum 411B) (Fig 395)

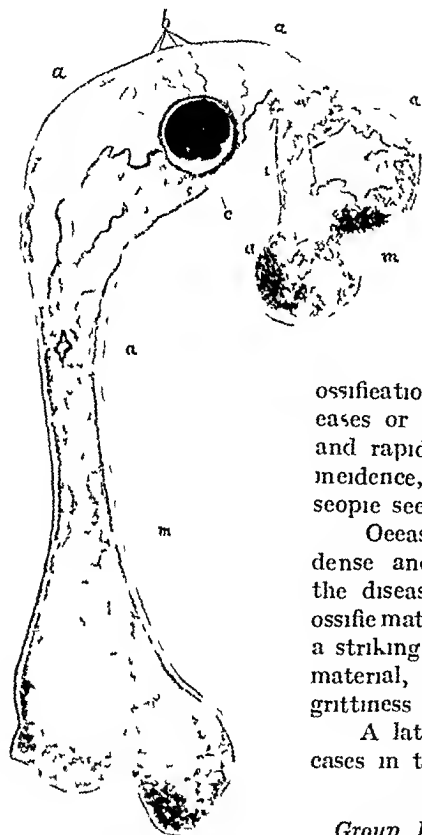


FIG 394—Femur from von Recklinghausen's case. (a) White fibrous tissue. (b) White porous bone formed within the fibroma. (c) Cyst with dark brown slummy material. (m) Several brown pimented spots strewn in the yellow marrow and the remains of the spongia of the epiphysis. (d) Region of impaction. (Reproduced from the LITE OF SURG 1914 II 9)

Case 8—This was the case of a man, age 53, in whom, during a period of great privation in early boyhood the first sign of the disease appeared in the tibia. The limb was amputated for sarcoma, which had led to spontaneous fracture of the femur. The abdominal glands were affected when the patient was last seen, and a fatal termination was evidently imminent.



FIG 395.—The tibia from *Case 7*. After Shattock and Leonard Little (*Spec 411 D, St Thomas's Hosp Museum*).



FIG 396.—Microscopic section from the lower end of tibia, *Case 8*. The fibrous tissue filling in the spaces between trabeculae of new formation and the absence of fat in the fibrous areas, are well shown. Note the sharp demarcation of the fibrous areas from fat marrow, and the long atrophic original trabeculae which mark the separation. Low power.

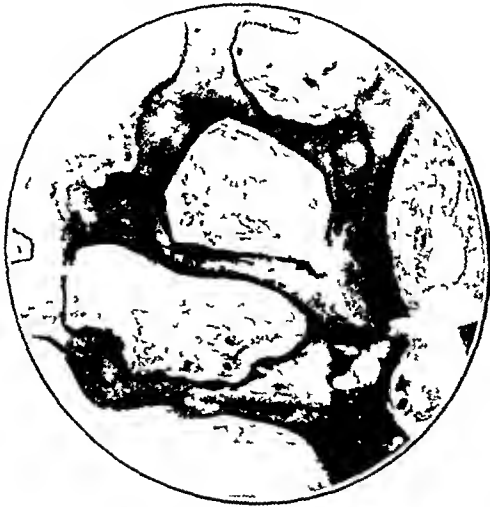


FIG 397.—A high power view from the same microscopic slide. Note the non laminated trabeculae and the giant cells.

The femur, the tibia, and the fibula were all extensively affected with osteitis fibrosa.

In addition to the gritty character of the fibrous tissue which replaced considerable portions of the cancellous structure of all three bones, masses or areas of very dense bone were present. These evidently represented the final stage of the disease. Some occupied the central medullary cavities, and had clearly been preceded by fibrous tissue. (Figs 396, 397.)

In these two cases (Nos 7 and 8), beyond slight bending there was very little alteration in the shape of the bones.

Case 9—Franklin and Edgecombe's case. The patient was a lady, age 65, who gave a history of a deformed tibia from the time she was two years old. Previous to

amputation she had been under observation for more than two years, and a swelling over the upper part of the bone appeared about the beginning of that time. It gave the egg-shell crackling sensation, and it was feared that malignant disease had supervened. The tibia was the only bone involved.



FIG 398.—Section from the chief fibrous mass in the tibia (Museum 711 W). It shows the bone formation taking place in fibrous tissue by metaplasia. $\times 30$

The other two masses of fibrous tissue lie amongst this bony fretwork, the spaces of which elsewhere are filled with fat. The latter is clearly a reversion from the connective tissue in which the new bone formation developed. The fat-marrow in the locular spaces is separated from the dense osseous septa by a fibrous membrane that can be lifted off the bone, which is seen to be dense and smooth and hard. It is probable that the bone in childhood would have presented an appearance very similar to that seen in Bilton Pollard's case (Case I). (Half the specimen is in the Pathological Museum of the Leeds University, and the other half in that of the Royal College of Surgeons, No 711 W.)

Group II — SINGLE CYSTS OF BONE

Attention has already been directed to cases of fibrous osteitis in which a considerable mass of fibrous tissue contains one or more cysts (Group II). As a rule the cyst is only a minor matter in comparison with the amount of fibrous tissue in which it forms. There are cases however in which a cyst appears to constitute the whole pathological condition. Such cases fall into Bloodgood's first and second categories, viz, those cysts in which there is no lining membrane at all and those in which there is a definite lining membrane.

At the first glance the specimen (see Fig 404) suggests a bone affected with osteitis deformans, owing to the bent shape, the uniform enlargement of the shaft, and the curious arrangement of bone in its interior. But there are at least three considerable masses of fibrous tissue present, and the largest $2\frac{1}{2}$ in long, occupies almost the whole sectional transverse area of the bone, reaching to the periosteum on the sides and in front. At its lower end it blends with a strong irregular fretwork of bone occupying the marrow cavity and gradually passing into the cancellous tissue of the lower end (Figs 398, 399).



FIG 399.—A portion from the same micro section as Fig 398. (L.S. Dr. H. Rodman, 1907)

composed of condensed connective tissue Yet even in the first group there is a zone of new connective tissue between the trabeculae of the bony wall

Is there sufficient justification for believing that these cysts originate in a pre existing area of fibrous osteitis?

Apart from parasitic cysts, and degeneration or hæmorrhagic cysts in growths, cystic formation in bone may result from (a) liquefaction of fibrous tissue (osteitis fibrosa) (b) liquefaction of cartilage (? chondroma, compare Case 6), (c) absorption of (necrotic) bone in such a manner as Nicholson has described as occurring in rheumatic arthritis (d) the presence of a simple serous cyst (?)—at present this is hypothetical

In the first three, a zone of young connective tissue forms at the periphery We may suppose that it represents Nature's attempt to isolate the disease The presence of such a zone would not by itself appear to justify the diagnosis of osteitis fibrosa cystica That term should be reserved for those cysts that result from degeneration of the fibrous tissue which has replaced a portion of the bone structure

Such an origin may be inferred when a cyst and a separate fibrous mass occur in the same bone (Case 3), or when a cyst occurs as part of the generalized disease, or when small patches of fibrous tissue are found in the immediate vicinity of the cyst

Case 10—Bland-Sutton's case of cyst in the humerus is an example of the last mentioned variety, showing a small mass of fibrous tissue in the bone which intervenes between two portions of the cystic cavity, and establishes its real nature to the naked eye (1637 D, R C S Museum Also Fig 30, Elmslie's paper¹⁸)

Also the diagnosis might be considered proved by such a microscopic section as Bloodgood shows¹⁹ in Fig 23 in his paper (p 161, Sower's case) It is taken from the bony wall of a cyst in the shaft of a humerus in which there was an unusually thick lining membrane It is particularly instructive Fibrous connective tissue is seen filling the spaces between the trabeculae, and in it are numerous small cyst formations which suggest that confluence of similar cysts is the explanation of the large one Case 10 is typical of a form of osteitis fibrosa cystica which is probably not very rare The majority of such cases occur in the upper ends of the humerus, femur, and tibia, and in the cancellous end of the diaphysis not far from the epiphyseal disc, but other bones and other situations are not exempt The cyst is usually conspicuous in a skiagram

Such cysts are liable to be brought to light when an injury results in a partial or complete fracture, and consequent disability They contain a serous fluid which may be yellow and clear, or chocolate coloured from old hæmorrhage, and in some cases cholesterol crystals are present Cultures are usually sterile

HISTOLOGY

The histology of osteitis fibrosa is, in the main, fairly constant

1 The ordinary bone marrow is replaced by a dense vascular connective tissue This is composed of fusiform or branched cells with outrunning processes It may assume the appearance of ordinary fibrous tissue and even show a whorled arrangement

2 All the fat disappears

3 The osseous framework has given place to this connective-tissue development From the thoroughness of its removal it may be surmised that it vanishes with unusual rapidity but there is very little evidence of the way in which it is destroyed

4 Throughout this connective-tissue replacement numerous scattered foci of new bone are forming These foci grow into trabeculae which in turn coalesce and form a network Eventually they develop into sclerosed masses of bone, whose fibrous character is often very apparent under the microscope (Fig 490)

* A similar change in the marrow occurs in some other diseases This is so in osteomalacia, in rickets and in osteitis deformans but each of these diseases has distinguishing histological features In osteomalacia identical ossific foci in the connective tissue may sometimes be seen but whilst new bone formation is the rule in osteitis fibrosa it is rare in osteomalacia except in callus Again, whilst the new bone trabeculae in osteitis fibrosa are usually completely calcified and only occasionally assume the osteoid form, in osteomalacia they are always incompletely calcified and composed of osteoid tissue

In a microscopical section from the fibrous area in a case of *ostitis fibrosa*, ossific points or small trabeculae may be seen, often in considerable numbers, scattered irregularly through connective tissue. In some parts of the section they constitute its most conspicuous feature, in others they may be rare or altogether absent. The new trabeculae are not laminated—certainly not in the early part of their growth—and the bone-cells are large rounded or triangular, and not flattened and stellate as in normal bone. At the periphery of the disease such new trabeculae may sometimes be seen based upon old laminated ones (*see Fig. 396*).

Ossification begins either by *metaplasia of small patches of connective tissue* (*Fig. 401*), or by deposit of calcareous granules round a connective-tissue cell in a matrix formed by the connective tissue itself. A group of a few adjacent cells, so altered, form a small calcareous mass and the fibrils of the connective tissue can be traced into its sides. At its edge cells

appear—as osteoblasts—in the spaces between these fibrils, and are gradually incorporated in the bone, which as it grows, assumes the characters and form of a trabecula, but without the normal lamination. Growth and resorption are active in connection with these new formations, especially where the connective tissue is very cellular—a row of osteoblasts and several osteoclasts may often be seen applied to the edge of a single trabecula, and the modelling process is evidently going on energetically. (It may result in laminated trabeculae.)

An intermediary stage of fibrocartilage in the ossifying process has been observed (Elmslie), but is very uncommon, and the formation of osteoid trabeculae, to which a reference has already been made, would seem to occur in cases in which a large amount of new bone has been laid down (*Cases 7 and 8*).

5 The origin of the cysts is not very clearly traceable in the microscopic sections. In certain cases there is a tendency for tracts of the connective tissue to pass into a state of necrosis, leaving only a framework of myxomatous tissue, the nuclei failing to stain. Such areas of degeneration are probably a first step to the production of a cyst, but they

are met with not only where cysts have already formed, but in other long-standing cases in which no such tendency has shown itself. The cyst contents are in most cases a pale yellow serum, which suggests that the process of softening arises from a liquefactive rather than a muemoid degeneration of the fibrous tissue. The section from Sower's case, previously mentioned, would seem to indicate that numerous minute cystic spaces first develop, and that a large cyst results from their coalescence.¹⁹

6 Finally it should be clearly understood that there is a marked absence of the small-celled infiltration met with in the more acute forms of inflammation.

THE CLINICAL ASPECT

The study of the different groups of cases enables us to follow not only the evolution of the affection, but also its clinical progress. The disease usually begins in childhood or during the growing period and if left to itself may last a lifetime. There is some reason to believe that it may become stationary or even go on to a spontaneous cure.



FIG. 400.—From the same case as *Figs. 401, 402*. Part of a patch of sclerod bone in the fibrous mass. An under exposed plate to show the architecture of the bone but failing to produce the connective tissue marrow which fills the lacunar spaces.

Three types may be distinguished —

- 1 A limited local deposit, frequently becoming a cyst
- 2 A more diffuse affection of a single bone involving the whole or a considerable part of the diaphysis
- 3 A generalized form in which many bones are affected

The patient only comes under observation when the disease is pronounced, and for one of the following conditions —

- 1 An enlargement of the bone, not readily noticed unless the bone is comparatively superficial
- 2 Deformity due to bending of the affected bone or bones
- 3 Fracture, which may be partial or complete and often spontaneous

- 4 A limp caused by shortening

A fracture is a very common complication. Four cases, at different times, came under my observation at the Leeds Infirmary, three were of the generalized type, and all three had suffered from one or more fractures. One of these was a boy, age 12 (Gerald G). At a time when both arms were in splints for fractures of each humerus, he was trying to raise a door latch with his head when both his femurs broke 'with a crack'. All the fractures united, and it fell to me to remove three

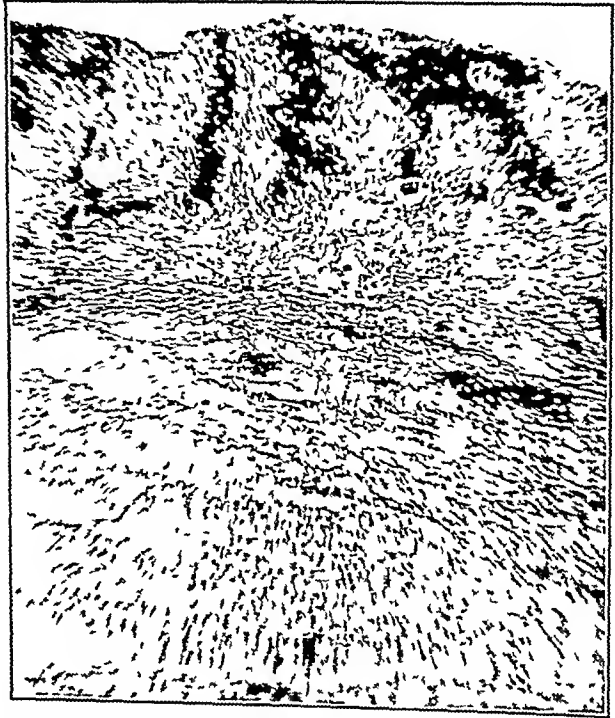


FIG 401.—From the surface of the same section as Figs 400 402. Showing the formation of bone by metaplasia of the connective tissue. Figs 400 401 402 are from different parts of the same microscopic section. (By Dr. O. C. Gruner.)

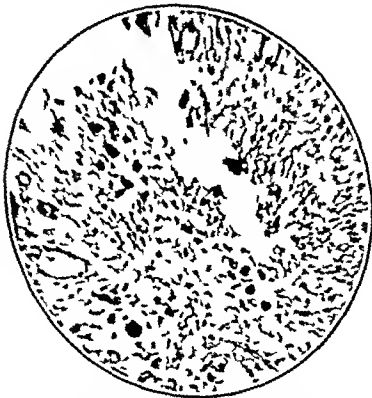


FIG 400.—From Littlewood's case of osteitis fibrosa of lower jaw. A part where many test cells were grouped together. (Leeds Med. Museum.)

trophone with a very large circle. The histology was that of osteitis fibrosa.

myelomata from his upper and lower jaws. He died two years later of 'heart failure', and during the last ten months was bedridden.

In some cases the disease is of such a mild character that a sufferer may be able to continue in active work throughout a fairly long life without suspecting that he is the subject of a progressive disease, until laid up by a late and probably final complication. In other cases a patient may welcome amputation to be rid of the incubus of a deformed and useless limb.

The long bones are most commonly attacked, but the skull is also frequently the seat of disease. Under the somewhat vague appellation of leontiasis ossium Victor Horsley described five cases of hyperostosis of the frontal bone. Four of them were almost certainly examples of osteitis fibrosa.

My fourth hospital case, a girl, aged about 16, had a swelling on the frontal bone which was so small that its removal was accomplished by a thin cut through the bone with the greatest ease.

The ossifying process that goes on in the fibrous tissue may be regarded as Nature's attempt to produce a cure, but it rarely comes to anything, for if the bone is made firm at one part, the disease is usually in active progress at another.

Fibrous osteitis is occasionally complicated by tumour formation. The common tumour is giant celled myeloma. This might be expected, seeing that the disease involves the medulla and is often marked by considerable giant cell development (Fig 402). Malignant disease may also supervene and terminate life. An abnormal tissue in a state of ill-regulated activity for many years is an obvious predisposing cause of such a complication.

In Case 8 a spindle sarcoma caused spontaneous fracture. The connective tissue could be traced till it merged in the growth and a nodule in the adjacent muscle was "composed of a similar spindle-celled growth supported in a connective-tissue basis identical with that seen throughout the three bones."



FIG 403.—From a section taken from Howship's case of osteomyelitis (Spec 739 R.C.S. Museum). On the right is seen fat infiltrated with recently effused blood. The trabeculae are completely decalcified except for a very small fragment (shaded deeply) in the lower part of the omega-shaped trabecula. The whole cortex is very thin, the periosteum thin, immediately on the left of the two isolated trabeculae on the left side and just failing to be included in the drawing. The breaking up of the decalcified substance and its metaplasia into fibrous tissue is well shown in the upper part of the drawing. (Drawn with camera lucida by J. P. Ford.) See footnote.

THE PATHOGENESIS

The disease includes the removal of a tract of osseous tissue, and its replacement by fibrous or connective tissue in which ossification takes place in an attempt to repair the damage.

Why is the bone removed?— Possibly because some change has occurred in it which makes its removal necessary. The most probable change is an impairment of its vitality to such an extent as to cause its death or render it incapable of recovery.*

How can such a change in a bone's vitality be caused?

In the suppurative inflammations which end in necrosis, bacterial toxins play a considerable part. Not only do they excite the inflammation which kills the bone but they exert a harmful influence upon the bone and its soft tissues before the blood supply is cut off. Owing to the intensity and rapidity of the process, this latter influence is of very little moment.

In tuberculous inflammation toxic influence no doubt prepares the way for the more ready disintegration of the bone trabeculae by tuberculous granulation tissue. But in this instance it is more easy to appreciate the action of

* *How is the bone removed?* It is likely that removal is accomplished in one of two ways: (1) By the removal of necrotic trabeculae by giant cells in the way described by Nicholson (*supra*) or (2) By some such process as that shown in the accompanying drawing (Fig 403). This is from a case of osteomalacia (No 739 R.C.S. Museum) and shows fragmentation and disappearance of osteoid tissue. It will be noticed that there are no giant cells taking part in the process.

the poison In both these conditions the toxins are produced locally, and it is easy to infer their formation because the presence of micro-organisms can be demonstrated

But osteitis fibrosa is clearly not dependent on a local micro-organismal growth Nevertheless, by analogy, we may suspect that the preparatory changes in the bone which necessitate its removal are also caused by toxins In that case the poison is carried to the part by the blood-stream

The toxic substances may originate (1) From micro-organisms, (2) From tissue metabolism (compare CO_2), or (3) From intestinal sources

It is highly probable that a toxæmia capable of producing the damage may, in different cases, be derived from organisms of different kinds, or be of metabolic or intestinal origin It is even possible that the source may vary at different times in the same individual It is important that we should realize that toxins are not necessarily specific in the same sense as pathogenic micro organisms But, besides toxins, another factor comes into play, viz *the vitality of the tissues, and their ability to resist toxic influence* It is a matter of common knowledge that the power of resistance to infection by micro organisms is possessed by different individuals in very different degrees, and that even families may show proclivities to certain forms of disease in consequence But we may go a step further, and recognize that there is a tissue resistance to toxic influence, and that one organ or tissue in the individual may show it in less degree than the others This again is well known Such an organ or tissue constitutes a 'locus resistentie minoris', and its resistance may be broken down by toxins arising in different ways

This may be illustrated by an example A transient toxic nephritis (hematuria, etc) was in the first instance excited by a septic infection, a second time by an influenzal infection, and a third time by an intestinal one

The toxins may or may not have the same chemical composition, but they certainly have irritating properties which may fail to injure the tissues as a whole but may select and influence the one that has least power of resistance Moreover, the usual

circumstances that depress the vitality of tissues may cause such an one to succumb, though it may have resisted successfully till exposed to them If this reasoning is correct, it follows that the onset of osteitis fibrosa may be explained on the supposition that, in certain individuals, an injurious influence is exerted upon the bones by toxins, which may be derived from one or more of various sources, that such toxins pick out the bones because they happen to be the tissue of least resisting power in the particular individual,

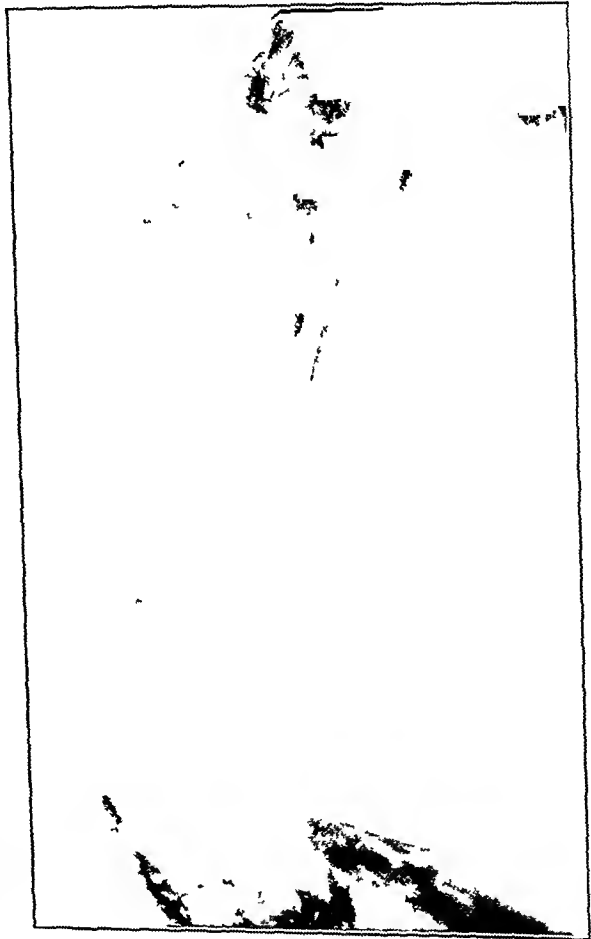


FIG 104.—Skigram of tibia from Case 9 Frankling and Edgecombe's case A partial fracture is seen at the upper part The pale area below is the main fibrous mass Below that a network of bone filled in with fat and occupying the central canal is indicated Still lower other fibrous masses above and below more indications of bone network, can be recognized

and that in many susceptible people they would fail to produce any obvious effect at all if some depressing influence (e.g., a fracture) had not lowered resistance still further, and determined the point of assault.

Little is known of the precise way in which the osseous tissue is affected by the toxins. The main mass of bone in a diseased area has disappeared before opportunities for microscopic investigation occur. But in trying to appreciate what takes place as a result of their action, we may picture to ourselves that both bone trabeculae and marrow are affected, but that the trabeculae, composed of a calcified matrix with only a few cells in its substance, are more vulnerable than the marrow. The influence that may damage the trabeculae beyond the possibility of recovery may excite reaction in the marrow. The tissue that is produced by this reaction is probably active in removing the moribund trabeculae with or without the aid of giant cells. At the first stage of the process with which we are familiar there is found, substituted for the bone and its marrow, a uniform tract of connective tissue exceptionally well supplied with blood-vessels. This tissue is osteogenic, and in places the beginnings of a new bone-formation are to be seen. Subsequent progress consists in a steady advance of irregular ossification. Resorption accompanies it, but whether this process is simply adaptive and healthy, or a removal of parts of the new bone brought under the influence of a continuous supply of toxins, it is impossible to say.

At last, in parts of a diseased bone the natural termination of the morbid process may be reached. The new bone becomes dense and sclerosed—in some places forming masses, in others strong septa—and the remaining connective (osteogenic) tissue reverts to adipose medulla (Fig 404).

These various changes point to an attempt on the part of the body to remove a portion of the framework of a bone which has been incapacitated beyond repair, and to replace it by a fresh development. When this has been accomplished, Nature demobilizes. The actual process by which the substitution is accomplished is, by most authorities, regarded as *inflammatory*, in spite of the fact that ordinary inflammatory cells are never in evidence. This view has been justified on the ground that "the new material has the loose connective-tissue structure of inflammatory new formation" (Report of the Committee of the Pathological Society on Morbid Growths. Messrs Shattock and B. Pitt's case). If the interpretation of the nature of the morbid process which has been suggested is correct, the disease would certainly comply with the conditions laid down by Burdon Sanderson, viz., "Inflammation is the succession of changes which occurs in a living tissue when it is injured, provided that the injury is not of such a degree as at once to destroy its structure and vitality."

TREATMENT

This should naturally concern itself in the first place with the cause. Any existing focus of sepsis should be removed. In a jaw case which I had the opportunity to investigate at a considerable interval after the removal of septic teeth, there was definite improvement and the hypertrophy had come to a standstill.

The dietary of these cases opens up a wide field for observation and experiment. There is good reason to believe that the pathogenesis of such diseases as rickets, osteomalacia, osteitis fibrosa, and osteitis deformans is allied. There is evidence that diet influences toxin production. It is not unlikely that Nature provides the antitoxin ready to our hand if we can only recognize and identify it. It matters not whether we call it antitoxin or 'vitamin'.

Tubby²⁰ records the case of a medical man who suffered from osteitis deformans. It is very significant. The patient attributed his improvement to the adoption of a diet rich in proteins and very sparing in carbohydrates, and the whole malady entirely to error in diet—viz., to lack of protein mainly, but partly to excess of starch. Eating potatoes never failed to produce a return of his pain.

Any measures that will assist in increasing the resisting powers of the patient, or prevent exposure to depressing influences, are of course indicated.

Various surgical procedures have been adopted in suitable cases. When a fibrous mass has been sufficiently local, its removal seems to have been justified by results. Bloodgood is of opinion that, if an osteotomy is required, curetting should be combined with it. Cysts have been curetted and packed, or filled in various ways, and the portion of bone affected by the disease has been excised and the gap dealt with. Hæmorrhage is the real danger in curetting operations when the disease is extensive, and fatal cases have been recorded. In some cases amputation may be advisable as an operation of expediency, a malignant complication of course renders it one of necessity.

OSTEITIS FIBROSA IN GOATS

In the Pathological Society's Transactions (1889, vol. xl, p. 449) W. G. Spence has given an account of a disease met with in goats. The affected animals were drawn from South London, where they had been kept for milking, consequently females predominated.

Before they were attacked by the disease they were fat, and their coats were smooth. Their diet had been hay, corn, cooked vegetables, and grubage, and they differed from goats fed on their natural food—hay and coarse upland grass—in that the latter are usually thin and have staring coats. Their ages varied from two months to three years.

The disease manifests itself by symmetrical swellings of the mandible, which gradually increase until the mouth cannot be closed, and death results from starvation, owing to the arrest of rumination in consequence of the immobility of the jaw. With the exception of changes in the upper and lower jaws, no others are perceptible, as a rule during life.

The disease runs a rapid course, death supervening from one to two months after the jaw swellings have become evident. The enlargements are produced by a new formation of homogeneous appearance and firm elastic consistency which replaces the original bone. They are most noticeable in the lower jaw, and involve particularly the angles and ramus, gradually shading off in the body. The upper jaws may also suffer, but to a less extent.

The swelling when cut across, is solid throughout of a pinkish white colour and spicules of bone can be felt to grate under the point of the knife. Both the skull and the long bones may also be affected, and become infiltrated by the new tissue. In one case the upper end of the tibia was replaced by the same pinkish-white growth as that in the jaws. Even enlargements of the bones may result and they can easily be cut with a knife. After maceration the bone is extremely light of the texture of fine sponge and may fall to pieces during the process. There is no evidence of rickets, and the epiphyseal lines are healthy. The changes in the jaw are said to begin in the interior of the bone about the tooth sockets. An upper jaw in the Royal College of Surgeons Museum (715D) (Fig. 405) shows a patch of new formation embracing the roots of a healthy tooth but this is separated by cancellous tissue from another patch. In specimen 366C in the St. Thomas's Hospital Museum, the bulk of that part of the swelling involving the body of the jaw intervenes between the erupted milk



FIG. 405.—Osteitis fibrosa in the upper jaw of a goat (Spec. 715, P.C.S. Museum). A mass of fibrous tissue is shown embracing the roots of a tooth and another mass adjacent to the nasal fossa. Possibly they are portions of a single mass; appearance is separate ones owing to the point at which the section was made.

teeth and the unerupted permanent ones, which are displaced downwards close to the lower border of the mandible—all the teeth are healthy

There is no true capsule to the new tissue formed in this affection. The morbid material merges with undestroyed bone, or extends to the periosteum, or blends with the peripheral bony layer which intervenes between it and the periosteum. In the St Thomas's specimen already referred to numerous small cysts the size of a pin's head are scattered throughout the mass, and these are larger and more crowded in the immediate neighbourhood of the embedded permanent teeth. Similar cysts can be seen in other specimens. A macerated lower jaw (715F, Royal College of Surgeons Museum) shows the interior of the swelling to be largely filled with a spongy or finely granular and excessively friable osseous material, which easily separates in powder. Obviously an osseous trellis work permeates the new formation at some period of its development. On the other hand, a sagittal section through the right femur of a goat, age 3 years (715II, Royal College of Surgeons Museum), shows only a thin compact shell with very slight remains of a rarefied cancellous tissue adhering to its inner surface. In the recent state it was filled with soft material.

Histologically the new formation (studied in the St Thomas's Hospital specimen) consists of a matrix of close connective tissue in which lies a network of osteoid or imperfectly calcified trabeculae. It is impossible to distinguish it from a section of fibrous osteitis in which the new bone formation is of an osteoid character, or from a section of callus formation in osteomalacia. There can be no doubt that the bone condition is osteitis fibrosa, and there is nothing to show that it has any connection with rickets. But the fact that in goats the jaws are specially selected for attack, though the teeth show no sign of disease, suggests that the act of rumination is probably a predisposing factor.

In its early stage the disease may be associated with a joint condition. Several joints may be affected and they contain darkly-stained synovial fluid. The synovial membrane is swollen and gelatinous, with dark hæmorrhages into it, and the cartilage may be eroded in spots and the bone exposed. The affection, however, is so slight that it may be overlooked, and the swelling which denotes it may disappear, leaving only slight traces behind. It is not clear how this peculiarity of the goat's disease is to be interpreted.

The author takes this opportunity to acknowledge how greatly he is indebted to Professor Shattock, not only for his assistance in placing much pathological material at his disposal, guiding him through the pitfalls of histological investigation, and directing him to reliable sources of information, but for much kindly criticism and other help.

He also desires to express his obligation to Dr O. C. Gruner and Dr G. H. Rodman, whose beautiful photomicrographs materially add to the value of this paper.

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CHRONIC DUODENAL ILEUS

By SEYMOUR BARLING, BIRMINGHAM

THE condition of acute gastroduodenal ileus, the so-called idiopathic dilatation of the stomach, which may arise as a post-operative complication after any surgical procedure, or even occasionally as a primary condition, is well recognized, but the closely related chronic condition has had little attention paid to it in this country till Wilkie's¹ paper focused attention on the matter. The condition is commoner than the literature on the subject would lead one to suppose, and both from the symptoms directly due to it, and from the part it plays in the production of other abdominal disorders, is worthy of further study.

Chronic duodenal obstruction may arise from a number of causes—some congenital in origin, such as partial atresia, ring pancreas, or excessive duodenal fixation by adhesions, in other cases neighbouring inflammations or growths may act by invading the duodenal wall or embarrassing its musculature, by kinking or direct pressure. In both acute and chronic ileus a most frequent cause is obstruction by the pressure of the mesentery and its contained vessels as it crosses the viscus. As the superior mesenteric artery and vein pass downwards across the third part of the duodenum, they normally produce a slight narrowing of the lumen at this point, in cases of visceroptosis or in abnormalities of the mesentery or the origins of these vessels, the duodenum may be excessively compressed near its termination.

In the adult the duodenojejunal junction is the point of transition of a relatively fixed portion of the alimentary canal into a mobile portion. Furthermore, the degree to which this process of fixation of the duodenum occurs in the embryo is one which varies within wide limits—it is especially towards the more distal parts of this length of bowel that fixation occurs.² The fixation of the duodenum occurs primarily by fusion of the mesoduodenum with the mesocolon and the structures lying in the neighbourhood of the right kidney; in addition, other secondary adhesions occur, especially around its terminal part. The frequency and variation of the peritoneal pouches around the duodenojejunal junction are explained by the irregular occurrence of these adhesions. Again, the neighbourhood of the third part of the duodenum is the pivot around which intestinal rotation occurs, rotation which brings the caecum across from the left to the right side of the body and carries the root of the mesentery of the jejunum and its contained superior mesenteric artery and vein athwart the duodenum near its junction with the jejunum. Embarrassments thus produced by abnormal adhesions and by the presence of peritoneal pouches into which herniation may occur, may be additional factors in rendering this part of the bowel vulnerable to obstruction by the crossing of the mesenteric vessels, themselves liable to great variation in tension, depending on the length of the mesentery, posture, and the presence or absence of distention of the small intestines.

Compression of the third part of the duodenum by the mesenteric vessels is a cause of acute gastric dilatation as suggested by Rokitsky³ in 1849. In 1889 Glenard⁴ regarded the dilated stomach as dragging on the duodenojejunal junction and so causing chronic obstruction at this site. Albrecht⁵ reported cases of chronic obstruction in 1899 due to flattening of the duodenum beneath the mesenteric vessels, and demonstrated the obstruction by experiment. Robinson,⁶ in 1900, pointed out the importance of mesenteric obstruction of the duodenum as a cause of gastroduodenal dilatation, and gave an account of the clinical symptoms and autopsy findings in chronic cases of this nature.

From this time onwards with increasing opportunity of studying the relationship of the parts in the living many papers have been devoted to the subject—notably by

Conner,⁷ Bloodgood,⁸ and Codman.⁹ The last drew attention to the local and general toxic effects produced by duodenal stasis and its action as a causative factor in the production of chronic duodenal and gastric ulcer, cholelithiasis, and pancreatitis.

The studies of Wipple,¹⁰ Sweet,¹¹ Ellis,¹² and many other workers as to the cause of death in acute intestinal obstruction and the production of toxic substances in the mucosa of the duodenum and jejunum under conditions of stasis, throw further light on the etiology of acute and chronic gastroduodenal ileus. The toxin is a systemic poison, causing the rapid collapse and death in the acute cases, whilst in the chronic ones its local action is related to the causation of chronic gastric and duodenal ulceration and gall-stones.

Kellogg¹³ reviews the whole subject in 1921. He gives particulars of 41 personal cases, and attaches an extensive bibliography. He performs the operation of duodenojejunostomy in suitable cases. It is noteworthy that of his 41 cases no less than 22 had been operated on previously, of these, 8 had had gastro-enterostomy and 12 appendicectomy, presumably without relief of symptoms.

It would seem probable that in both the acute and chronic ileus a primary underlying obstructive cause is present at the site of the crossing of the mesenteric root, but that, in addition, in the acute cases a secondary toxic factor is added which overwhelms the patient and is responsible for the grave collapse seen in these cases, and also for the altered conditions of the stomach musculature and secretion. Such toxic symptoms are present, though in a greatly lessened degree, in the chronic cases where they are manifested by headache, dizziness, malaise, and distaste for food. Alterations in the gastric and duodenal mucosa, as shown by hæmatemesis and ulceration, and malnutrition from stasis, are the chief local effects seen in the chronic cases.

Of the 7 cases on which this paper is founded, in 5 the symptoms appeared to be due to obstruction at the crossing of the mesentery. In these the hypertrophied and dilated stomach, widely patent pylorus, and dilated duodenum, ending abruptly at the site of the crossing of the mesenteric vessels, presented a striking picture at operation. In one of these cases there was a large chronic ulcer on the lesser curve. In the other 2 cases the obstruction was produced by the contraction of tissues lying in proximity to the duodenal wall. The symptoms in these cases were so similar to those presumed to be due to mesenteric constriction that they serve to strengthen the contention that the latter is a true cause of mechanical obstruction, and the primary condition accounting for the symptoms. In one of these cases (*Case 6*), the enormous dilatation of the stomach on admission, and the grave collapse and copious vomiting, closely resembled the condition seen in acute gastroduodenal ileus.

Symptoms—The symptoms of digestive disturbance produced by chronic duodenal stasis usually show a gradual ungracefulness over many years, with a tendency to acute exacerbations induced by dietetic indiscretions, posture, or even chronic constipation. During an attack epigastric pain is present, vomiting is common and often copious, and flatulence and epigastric distention are very marked.

Absorption from the toxic contents of the duodenum may cause headache or dizziness, or give rise to an icteric tinge of skin and conjunctivæ. Loss of weight may be considerable in the later stages of the condition. In some cases blood has been noticed in the vomit.

Patients with this trouble have often been submitted to surgical operation before the condition is recognized, the trouble being variously diagnosed in the appendix, the stomach, the duodenum, or the gall-bladder, as the symptoms may mimic disease of each of these organs very closely. Not only so, but the ileus may accompany chronic gastric and duodenal ulcer and gall stones as a causative factor. The relationship of the onset of pain to the taking of food is not usually as definite as it is in cases of uncomplicated chronic gastric or duodenal ulcer. Nor is the pain so severe, being rather of the nature of intense flatulent discomfort. Vomiting is apt to be more copious and more frequent than is usually to be found with these conditions except when they are associated with a high degree of pyloric stenosis, furthermore vomiting does not give that relief to pain that it does in chronic gastric ulcer. The regurgitation of bile through the open pylorus that is

found in duodenal ileus is a very important diagnostic point as it is nearly always absent in the vomiting of pyloric stenosis. During the attacks there are often nausea and distaste for food though appetite may be normal at other times.

To sum up, the clinical history simulates to some extent that found in chronic inflammation of the appendix and gall-bladder, or chronic gastric or duodenal ulcer. The pre-operative diagnosis in the 7 cases recorded in this paper was accurate in 3 instances, in the 4 other cases the diagnosis was pyloric stenosis, 2 gall-stones and chronic gastric ulcer 1 each. One of the cases had been operated on for chronic appendicitis before coming under my care.

X-ray Diagnosis—Screening the patient after a barium meal may be of great assistance in making a diagnosis, as duodenal stasis and distortion may be definitely seen in some cases. In 2 cases out of 7, accurate diagnosis was made on this observation. At other times however the x-ray picture is not so clear and in *Case 1* the whole of the meal was held up in the stomach at the end of four hours and the diagnosis of pyloric stenosis was consequently made. One other case had the large stomach with deep waves and a high degree of stasis suggestive of pyloric stenosis but in this patient the condition was diagnosed on the great epigastrie distention and copious vomiting of bilious material. In two of the cases there was some delay in emptying the stomach and some degree of pyloro-duodenal deformity. One case was reported as normal. Repeated observation of cases in which the symptoms are intermittent may be necessary to establish a diagnosis, and it is especially necessary to observe the duodenal loop immediately the meal enters the stomach and before the picture is obscured by jejunal filling, or the presence of an enlarged stomach in front of the loop.

Chemical Investigations—In the course of investigating a number of cases presenting gastric symptoms by means of the fractional test meal, Dr T. L. Hardy has made observations on three of my cases of duodenal ileus. In one the quantity of fasting juice, the

acid response, and the rate of emptying were normal. In a second case there was hyperacidity in the early stages, with efficient neutralization later, and a final rise to high values when the stomach was nearly empty. In a third case (*Fig 406*) it was possible to make the diagnosis of duodenal obstruction with some confidence from chemical examination alone. Over a pint of dark-green turbid fluid, in which no starch was present, was extracted from the fasting stomach. All subsequent specimens contained much bile while starch was present at three hours in considerable quantity. The acid values showed nothing noteworthy.

The value of gastric analysis in these cases will clearly depend on the state of the pylorus.

In the third case the duodenal obstruction was due to periduodenal contraction, the pylorus was patulous, and the stomach shared in the dilatation. The method is likely to be of value only in the more extreme types of the condition.

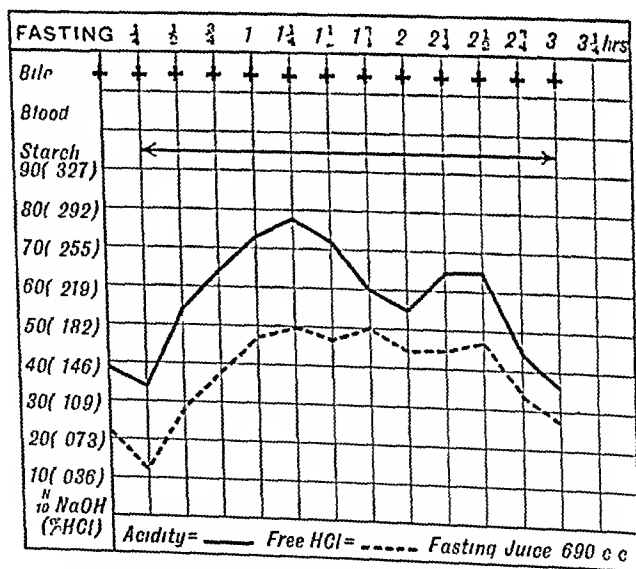


FIG 406—Fractional test meal in a case of chronic duodenal stasis (*Case 7*) due to cicatricial contraction involving the wall 1 in. from the pylorus.

ILLUSTRATIVE CASES

The following five cases present some of the salient features of the condition. In all the mesenteric vessels appeared to be the main cause of the obstruction.

Case 1—A man, age 35, who was serving in the Navy at the time of the onset of his illness and was otherwise healthy. His troubles commenced with severe colicky pain in the upper abdomen in 1916. The pain came on in attacks, was indefinitely related to food, starting from one to six or eight hours after a meal, and was accompanied by vomiting. He sometimes went as long as two months between attacks. In 1918 appendicectomy was performed, but though relieved for a while, in 1919 his attacks of pain and vomiting were again very severe. He vomited very large quantities, up to half a gallon. In November, 1919, he was again operated on at Chatham, adhesions being broken down and severe gastroparesis observed. In 1920 the old attacks recurred, and pain and vomiting were again severe. He was seen by me in 1921 for continuation of his pain and vomiting.



FIG 407.—Radiograph of Case 1 taken four hours after barium meal. None of the meal had passed the pylorus though it was widely open.

A ray examination (Fig 407) showed 'typical pyloric stenosis enlarged deep waves, all meal in stomach at end of four hours.'

In view of his condition and these findings, laparotomy was decided on, and to my surprise the pylorus was much broader than usual and admitted three fingers easily following down the duodenum it, too, was much wider than normal in its first, second, and third parts, and the dilatation terminated at the crossing of the superior mesenteric vessels, which stretched like a tight band across the bowel in its third part. Gastroenterostomy was performed, as the operation of duodenojejunostomy was unknown to me at that time and relief of the obstruction was urgently necessary, and gastroenterostomy would seem to afford it. The result was successful, for with the exception of two large vomits during convalescence, the condition has been entirely relieved, and the patient has put on four stone in weight in fourteen months.

Case 2—A woman, age 36. Symptoms of pain immediately after food, and frequent vomiting, for four years. Between 1919 and 1922 she had lost nearly three stone in weight, and was thin pale, and ill nourished on admission. She had localized tenderness to the right of the umbilicus, and in this area small peristaltic waves could be seen moving from left to right. The radiographic report stated that the stomach was small and quiet and there was slight pyloric deformity. In four hours one tenth of the meal was still in the stomach. At operation the stomach was normal except the pylorus, which admitted three fingers. The whole duodenum appeared dilated, for no apparent reason. On closer investigation, however, some adhesions were broken down which had cemented the dilated third part of the duodenum to the jejunum, and across the narrow isthmus of the bowel beneath these adhesions the superior mesenteric vessels were tightly stretched. Duodenojejunostomy was performed, and the appendix removed, as it was chronically inflamed. The patient made a satisfactory recovery.

It is interesting that in this case a large retroperitoneal pouch of peritoneum passed from the left side of the duodenojejunal junction for quite 2½ in upwards and to the right behind the duodenum. This, if distended, must have increased the obstruction caused by the vessels. It is possible that the attacks were induced by herniation of bowel into this pouch.

This patient was operated on a year ago. She has now lost all symptoms of indigestion except some flatulence and has recently given birth to a child. She has put on over a stone in weight.

Case 3—A woman, age 50. For three years she has had attacks of epigastric pain and vomiting, the attacks getting more frequent and severe. During the attacks she has epigastric and left hypochondriac pain of a colicky nature with frequent vomiting and great nausea and distaste for food. The vomiting does not relieve the pain, and the vomit contains bile. There is much flatulence. Between the attacks the appetite is good. There has been some loss of weight. Skrimger showed nothing abnormal in the stomach or duodenum. A preoperative diagnosis of gall stones was made.

At operation, the stomach and duodenum as far as the crossing of the mesenteric vessels were dilated and hypertrophied, and well marked pressure on the third part of the duodenum by the mesenteric vessels was observed when the finger was passed beneath them. Technical difficulty prevented the performance of a duodenojejunostomy as much bleeding ensued on attempting to mobilize the third part of the duodenum for its performance. A gastroenterostomy was therefore performed. This patient though better, still has nausea and I feel it would have been better if a duodenojejunostomy could have been performed and the dilated duodenum drained at its distal extremity.

Case 4—Male, age 33, who presented evidence of old ulcers in stunted growth, kyphosis, and a pigeon breast. This patient had symptoms of chronic indigestion of twenty years' standing, with pain, vomiting, and occasional hematemesis occurring in attacks. He was admitted during one of these, and was thought to be bleeding from a chronic ulcer of the stomach or duodenum, as he had suffered recently from dizziness and fainting, and had melena on admission. Vomiting had been more frequent recently, and did not relieve his pain. The vomit contained bile. A x-ray examination failed to indicate the presence of ulcer, but some degree of stasis was present in the stomach. The appetite and nutrition were both poor, and he suffered from much flatulence.

Operation showed a hypertrophied and dilated stomach. The pylorus was 2 inches broad, and the duodenum dilated to the crossing of the mesentery, the bowel being normal beyond. General visceroptosis was present. Duodenojejunostomy was performed. At the end of three months he is much improved, but not wholly comfortable after meals. Weight is increasing.

Case 5—Chronic duodenal stasis with chronic gastric ulcer.

Male, age 32, with a history of two months severe indigestion. Up to the time of onset, patient states, he was free from any digestive troubles. The onset was abrupt, with severe pain in the left hypochondrium, the pain coming on two hours after food, so that he was afraid to eat, though his appetite was good. Vomiting was frequent, especially at the onset of his illness, it relieved his pain, and he noticed that the vomit contained bile. His general nutrition was poor, and his mouth contained many septic stumps. Screen examination revealed a large chronic ulcer on the lesser curve (Fig 408) and a considerable degree of duodenal dilatation throughout the length of the viscus.

At operation a chronic ulcer was found on the lesser curve 3 in. from the pylorus, its crater



FIG. 108.—Radiograph of chronic gastric ulcer (A) in a case of chronic duodenal stasis due to pressure of the mesenteric vessels. Taken ten minutes after swallowing the meal. Obstruction is incomplete as some barium has passed the duodenojejunal junction. The whole duodenum was dilated but this was more evident with the screen at the moment of entry of the barium than is apparent in the picture (Case 5).

easily accommodated the tip of the index finger when the stomach wall was invaginated into it, the pylorus was broad, the first part of the duodenum was 3 in. across, and the dilatation was so great in the third part that this retroperitoneal part of the gut was easily brought out of the abdominal cavity on turning up the colon. The dilatation ceased at the crossing of the mesenteric vessels. The jejunum was anastomosed to the third part of the duodenum, and the ulcer was left alone in the belief that it would heal when the stasis was relieved. This patient is doing well, but it is too early yet to say if the ulcer has been permanently cured by the relief thus afforded.

The two following cases present in their symptoms a picture very like that observed in those recorded above, and may be considered with them. In both of them obstruction of the duodenum was produced by contraction of the periduodenal tissues in the one case by malignant disease, and in the other apparently by cicatrization secondary to some chronic adenitis in the neighbourhood of the viscera. The obstruction thus produced caused changes in the stomach and duodenum analogous to those seen in the cases due to mesenteric drag, though the development of symptoms was more rapid and lacked the characteristic intermissions of the latter cases. The close similarity between the symptoms seen in the two types of case affords, I think, strong evidence of the reality of the obstruction produced by tension of the mesenteric vessels where they cross the duodenum.

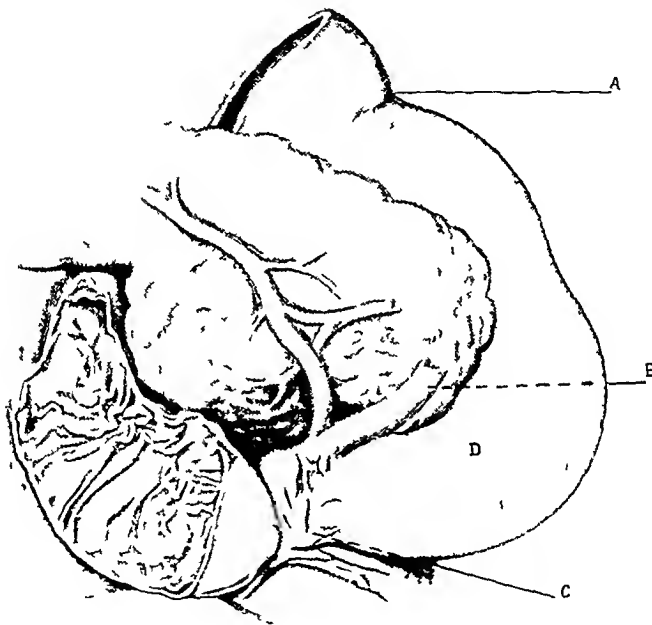


FIG. 409.—Duodenal obstruction by growth arising in the pancreas and invading the wall of the duodenum distal to entrance of common bile duct. Seen from behind (Case 1). A, Pylorus, B, Common bile duct, C, Growth, D, Distended loop of duodenum.

Case 6—A domestic servant, age 32 who had presented no symptoms of digestive disorder up to the time of her present illness, was sent urgently to hospital in an extremely collapsed condition. Epigastric pain and vomiting had commenced suddenly two months previously, and vomiting had been frequent and copious ever since, especially so just previous to admission, so that when first seen the patient was almost moribund from loss of fluid. The abdomen was distended as by a large low tension cyst, and this, and the presence of a succussion splash, led to the passage of the stomach tube, which drew off 6 pints of brownish fluid. The house surgeon who saw her on admission stated that the tube could be felt in the stomach through the thin abdominal wall. As low down as the brim of the pelvis. With daily lavage the patient improved and the vomiting ceased. Somewhat later, however, the vomiting again set in, and it was evident that if anything were to be done by operation it must not be delayed.

The radiographic report was as follows: "Stomach normal in size and shape, stasis in second part of duodenum—four hours, a fifth of contents was still in the stomach." The patient presented the appearance seen with acute gastroduodenal ileus—a feeble pulse, extreme lethargy, and cold

extremities—and although large quantities of fluid intravenously and subcutaneously improved her slightly, she was very feeble at operation. Laparotomy showed a stomach of moderate size with thick walls, the pylorus was broad and the duodenum very dilated. The dilatation ended at the middle line at a point which appeared as if the termination of the duodenum. Here a small hard mass of infiltrated tissue was present, and the front surface of the bowel was scarred over and contracted, the whole being not unlike a cotton reel in size and consistence. The exact nature of the obstruction could not be determined and so it was short-circuited by the rapid performance of a duodenojejunostomy.

The patient appeared little the worse for the operation but gradually faded out in the next thirty-six hours. Post-mortem examination showed a thick-walled stomach of moderate size, and great dilatation of the duodenum as far as the obstruction, where the bowel was embraced by a mass of cicatricial tissue (Fig. 409). Closer examination of the site of the obstruction showed that it was at or near the entrance of the common bile duct which was apparently lower down in the duodenum than normal. The tissue causing obstruction was mainly fibrous, but was of a spheroidal celled carcinoma in the fibrous mass indicated that a scirrhous growth of the pancreas was probably the primary cause of the obstruction.

Case 7.—Periduodenal fibrosis around the second part of the duodenum obstruction

A male, age 52, had sudden onset of pain and vomiting, which continued with increasing severity up to the time of admission fourteen days later. The pain was severe and unrelated to food and was epigastric in site. Vomiting was frequent and copious, and took place at indefinite times of the day and night, and the vomit contained bile. His general nutrition was good, though he stated he had lost three stone in weight in the last two years. He had much epigastric distention, and the outline of a hypertrophied and dilated stomach could apparently be felt coming low down in the epigastrium. X-ray examination showed an enlarged stomach with deep waves, and at the end of four hours a third of the meal was in the stomach. A diagnosis of duodenal obstruction was made on clinical signs.

At operation, the stomach, and the first and second parts of the duodenum, were found to be greatly hypertrophied and dilated, the pylorus easily accommodated three fingers. There was no dilatation of the third part. The dilatation ended 3 in. from the pylorus in a cicatricial ring, from which a small portion of tissue was removed for histological examination. The pathological report on this showed it to be dense fibrous tissue only, and there was no evidence of its origin.

As the obstruction was so high up it was decided to do a gastro-enterostomy. To bring a loop of jejunum so far across to the right, in order to do a duodenojejunostomy, would, it was feared, result in causing embarrassment to the function of the loop. Convalescence was marred by several bouts of vomiting in the first week, but otherwise the patient made a good recovery.

Cases 6 and 7 belong to a well-recognized but comparatively small group in which duodenal obstruction is caused by involvement of the second or third parts of the bowel in neighbouring inflammations or growths, and have only been mentioned because of the similarity of the picture they present to the cases of obstruction by the crossing of the mesenteric root. Of obstruction caused by the latter method the five first cases were well-marked examples, but it seems probable from observations in the course of performing laparotomy for other conditions that duodenal dilatation of lesser degree is not at all uncommon, and though in these cases the condition is not sufficiently marked to call for surgical relief yet it may be a factor in producing disturbance of function in the stomach, duodenum, or biliary tract.

The cases quoted were so well established that there can be no doubt they were wholly responsible for the patients' condition, and merit recognition as a clinical entity, definitely to be borne in mind when investigating a case of chronic indigestion. Similarly, when performing laparotomy for this condition, a routine examination of the third part of the duodenum must be made after the examination of stomach, gall-bladder, appendix, and other possible sources of trouble.

Treatment—The relief that is afforded in acute gastric dilatation by lavage and posture suggests the possibility of using these measures in the chronic condition. Furthermore, it is possible that abdominal support by a suitable belt may be of value in relieving the splanchnoptosis so commonly found with the condition. In cases, however, in which the obstruction has reached a certain degree of severity, permanent relief can probably only be obtained by operation. In these cases the stasis is such that from time to time the hypertrophy of the stomach and duodenum fails to overcome the block, compensation fails, and hypertrophy gives way to dilatation. When this stage is reached, relief is permanently given by short-circuiting the duodenum in its third part into the jejunum,

close to its commencement This procedure was suggested by Barker¹⁴ in 1906, but the first case recorded in which it was carried out was by Stavely¹⁵ in 1908, full details of the operative procedure are given in Kellogg's paper¹³ The operation resembles technically a gastro-enterostomy, but is a little more difficult to perform, as the duodenum is retro-peritoneal and cannot be brought to the surface as easily as the stomach The dilatation of the horizontal part of the duodenum which is so noticeable in the established cases renders the operation easier than might be expected In Case 5, for instance, this part of the duodenum could easily be brought out of the abdomen, and the operation be performed with as much ease as the ordinary gastro-enterostomy In three cases a gastro-enterostomy was performed in the first, because the operation of duodenojejunostomy was unknown to me, in Case 3 an attempt to perform the operation of duodenojejunostomy was frustrated by considerable hæmorrhage from large veins in front of the dilated duodenum, whilst in Case 7 the obstruction was so high up in the duodenum that it appeared preferable to do a gastro-enterostomy

Convalescence in these three cases was unsatisfactory owing to vomiting, which was of the nature of that seen in the vicious circle after gastro-enterostomy, and was relieved by lavage Case 1 has done so well since leaving hospital that it would appear gastro-enterostomy may occasionally give permanent relief to the condition if the initial post-operative dangers of the vicious circle are tided over

Of the other two cases in which gastro-enterostomy was performed, one is definitely unsatisfactory, the other is relieved by the operation, but is too recent to enable one to judge of the permanency of the relief There is no doubt that duodenojejunostomy, by relieving the dilated bowel at its distal end, is the operation of election, whilst gastro-enterostomy is liable to be associated with a post-operative vicious circle

CONCLUSIONS

1 The condition of duodenal ileus may arise from pressure of the mesenteric root containing the superior mesenteric artery and vein on the third part of the duodenum

2 Whilst probably such obstruction is the chief predisposing cause of acute dilatation of the stomach, it may also give rise to chronic digestive disturbance, simulating chronic ulcer of the stomach and duodenum, or inflammation of the appendix or gall-bladder, and is sufficiently common to be recognized as a clinical entity

3 Apart from the direct effects produced by obstruction, duodenal ileus plays a part in the etiology of some cases of chronic gastric and duodenal ulceration and infections of the biliary tract

4 In many cases an accurate pre-operative diagnosis of the condition may be made by a careful consideration of the clinical symptoms and the results of a radioscopic examination

5 When performing laparotomy the condition must be borne in mind and sought for as a cause of chronic indigestion, and, in the presence of hypertrophy and dilatation of the stomach and duodenum with patent pylorus, the operation of duodenojejunostomy performed

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ABNORMALITIES OF THE RIGHT HEPATIC, CYSTIC, AND GASTRO-DUODENAL ARTERIES, AND OF THE BILE-DUCTS

By L. R. FLINT JUDS

HUMAN beings are singularly alike in their general anatomical construction yet when we come to investigate one particular region with more detail it is surprising how frequently we meet with variations of one sort or another. More especially does this apply to the vascular system and in no region more than to the liver. This is I think, generally appreciated by anatomists. There are however variations in the excretory ducts of the liver almost as frequently, and anatomists have not given the attention to this subject that a part so important from the surgical point of view, deserves.

I have made 200 dissections on post-mortem subjects of the vessels and ducts of the liver. The dissections were made as far as possible consecutively. There has been no selection of cases, and it is claimed, therefore, that the anatomical details given me a very fair representation of the state of the parts as found in the human subject and that the results of any one undertaking so large a number of dissections will be found to corroborate mine. In addition, I have paid particular attention to these parts during operations, but I have not included the results in the statistics, I shall refer to them later.

If we exclude the appendix the gall-bladder and its ducts more often call for operative treatment than any other intra-abdominal viscera, and a great deal of this surgery is very badly done if one may judge by the great number of cases one sees which require a second operation, mostly for reasons that should not arise if proper care and knowledge had been applied at the first attempt. Technically, gall-bladder surgery is much the most difficult of any abdominal surgery, and inadequate appreciation of the abnormalities of this region does not lessen the risks.

The arrangement of the vessels and ducts given is normal in the text-books of anatomy is shown in *Fig 410*. In my series of 200 cases, only 69 conform to this type. So frequent are variations that it is impossible to regard any one type as normal, the arrangement found in the 69 cases can only be described as the most usual one.

I will describe first the vessels as found in my series.

The Right Hepatic Artery—This arises from the main hepatic trunk in 158 cases, and to reach the liver passes behind the common hepatic duct in 136 (*Fig 410*), and in front of this duct in 25 (*Fig 411*). In 42 the right hepatic artery arises from the superior mesenteric artery (*Fig 412*), and always passes behind the common duct. In 7 cases there are two right hepatic arteries, one from the hepatic trunk and one from the superior mesenteric (*Fig 413*). In 2 cases there are two right hepatics both from the main hepatic, one passing in front of, and the other behind, the common hepatic duct. In 4 cases in addition to passing behind the ducts, the main hepatic or the right hepatic artery also passes behind the portal vein (*Figs 414 and 432*).

I am aware that the right hepatic artery very occasionally arises from the aorta, the right renal, the gastric, or the inferior mesenteric artery but there are no instances in my series.

The right hepatic artery, as it crosses the bile-duct in 25 cases—especially when crossing low down near the junction of the cystic duct—is liable to injury during cholecystectomy. There are two other variations which render it even more liable to an accident—

1 In 8 of the cases, all in elderly people, the artery is tortuous and projects forwards to the right of the common hepatic duct, something like the hump of a caterpillar's back during progression (*Figs 413 and 425*). From the summit of this hump may arise the

DIAGRAMS ILLUSTRATING VARIOUS ABNORMALITIES IN THE ARTERIES AND BILE-DUCTS MET WITH IN GALL-BLADDER SURGERY

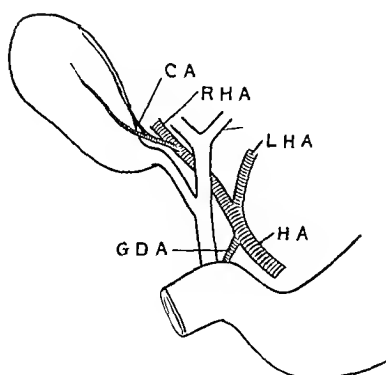


FIG 110

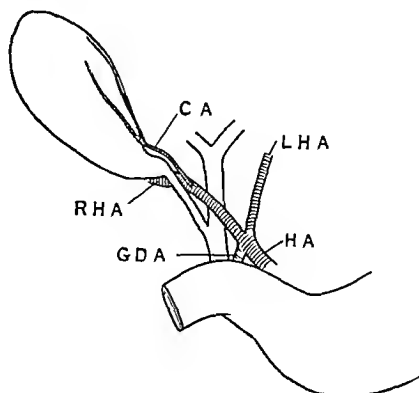


FIG 111

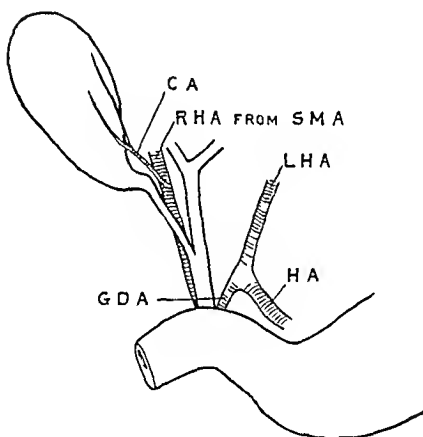


FIG 112

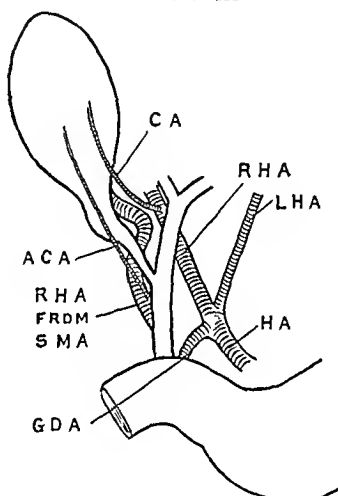


FIG 113

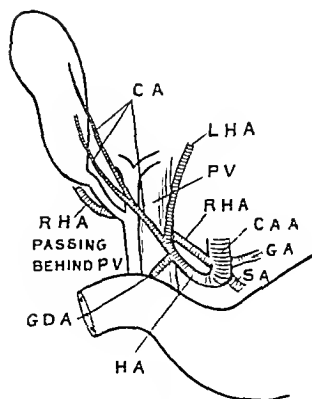


FIG 114

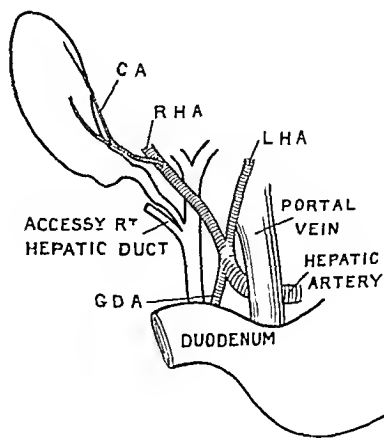


FIG 115

(C A) Cystic artery (G D A) Gastro-duodenal artery (H A) Hepatic artery (L H A) Left hepatic artery (R H A) Right hepatic artery (S M A) Superior mesenteric artery (A C A) Accessory cystic artery (P V) Portal vein (C A A) Celiac axis artery (G A) Gastric artery (S A) Splenic artery (R H D) Right hepatic duct (S P D A) Superior pancreatico-duodenal artery (C D) Cystic duct

cystic artery, and on pulling up the gall-bladder the projecting artery comes to lie at a level which is even a little anterior to that of the cystic duct. I have known this mistaken for an enlarged cystic gland, fortunately the error was recognized in time. I have no doubt, however, that it has been included in the clamp from time to time.

2 The right hepatic artery may run parallel and very close, to the cystic duct and the neck of the gall-bladder, almost suggesting a double cystic duct (*Fig 121*). It could very easily be included in the clamp applied to the duct.

The Cystic Artery arises from the right hepatic 196 times out of the 200, in 3 from the left hepatic (*Figs 418 and 434*), and in 1 from the gastroduodenal artery (*Fig 417*).

In 32 cases it passes in front of the common hepatic duct (*Figs 411 and 416*), and in 168 it arises just to the right side of the common hepatic duct (*Fig 410*) or behind it (*Fig 436*). The former is much the more common. This difference in point of origin has some surgical interest to which I will refer later.

Accessory Cystic Artery—In 31 cases there is an accessory cystic artery. There is therefore, a single cystic artery in only 169. Attention is not called to this in anatomy books. The accessory cystic artery arises from the right hepatic in 16 (*Figs 413, 419 and 420*), from the left hepatic in 3 (*Figs 421 and 434*) from the gastroduodenal in 11 (*Fig 422*), and from the superior pancreaticoduodenal in 1 case out of the 200 (*Fig 423*). In the last two instances it is liable to injury during the operation of cholecystotomy. When there was an accessory cystic artery I found that it invariably crossed in front of the bile-duets. Ignorance of the occurrence of accessory cystic arteries may be responsible for rather severe hemorrhage.

There are three other points of interest about the arteries.

1 In opening the common duct it is quite common to have an annoying hemorrhage from an artery which crosses the front of the supraduodenal part of this duct. Though a plexus of veins and arterioles is described on the surface of the duct I have seen no mention of this artery in the literature. Surgeons know it well, and I was curious, therefore, to discover its source. I am unable to give the frequency of its existence, as I did not begin to look for it from the first, but I found it quite often, it arises from the hepatic artery low down, or from the superior pancreaticoduodenal or from the gastroduodenal, and runs a rather tortuous course along the anterior surface of the duct (*Figs 425 and 428*). It may be the superior pancreaticoduodenal itself, when this vessel comes off higher than usual.

2 The gastroduodenal artery in a small percentage of cases forms a curve in front of the lower supraduodenal part of the common duct (*Fig 426*) and might be wounded in opening the common duct. Also the superior pancreaticoduodenal artery occasionally crosses the duct just above the level of the upper border of the duodenum (*Figs 423 and 425*).

3 Although this paper is chiefly concerned with the right hepatic artery, I should like to mention one point about the left hepatic. In my first 100 cases I dissected out this artery, and found it was quite common to have two left hepatic arteries. In 32 cases one artery came from the main hepatic and the other from the gastric, and in one case the only artery which could be found going to the left lobe of the liver came from the gastric. In doing a gastrectomy this vessel would almost certainly be divided, and troublesome bleeding might occur from the distal end.

The Bile ducts—The second part of this paper deals with the bile ducts. According to the anatomical text-books, the right and left hepatic ducts unite in the portal fissure, or just beyond it, to form the common hepatic duct. This structure is from 1 in to 1½ in long. The cystic duct is from 1 in to 1½ in, and uniting with the common hepatic duct at an acute angle, they together form the common bile-duct which is about 3 in long. The length of the supraduodenal part of the common duct varies with the level of the duodenum and the point at which the cystic and common hepatic ducts join, the average length for this part of the duct is held to be about one-third of the whole length of the common duct.

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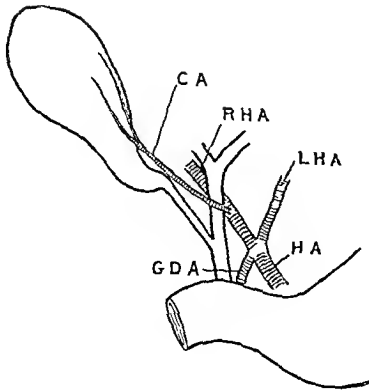


FIG 416

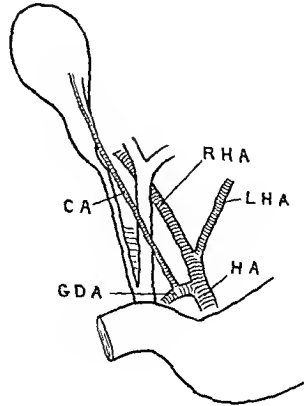


FIG 417

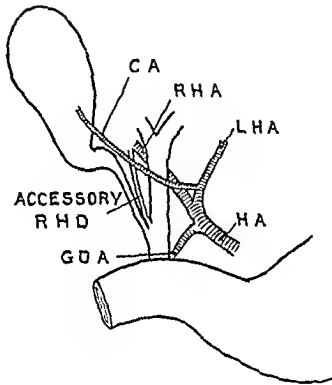


FIG 418

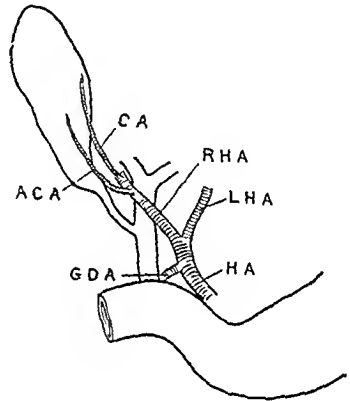


FIG 419

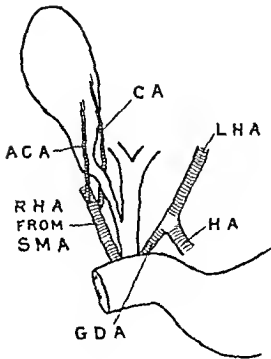


FIG 420

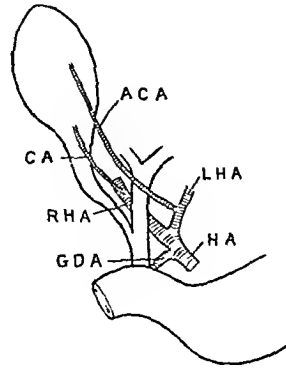


FIG 421

(CA) Celiac artery (GDA) Gastroduodenal artery (HA) Hepatic artery (LHA) Left hepatic artery (RHA) Right hepatic artery (SMA) Superior mesenteric artery (ACA) Accessory celiac artery (PV) Portal vein (CAA) Celiac axis artery (GA) Gastric artery (SA) Splenic artery (RHD) Right hepatic duct (SPDA) Superior pancreatico-duodenal artery (CD) Celiac duct

Now, though it is true that the cystic and common hepatic ducts do come together at such a point as to give an average of lengths as stated they do not unite here. Almost always they are merely bound together by fibrous tissue, and by dissection it is possible to separate them from each other for a few millimetres to as much as 2 in. or more. The most common point, according to my observations at which union actually occurs is within 1 cm. of the upper border of the duodenum (*Fig 117*)

In 28 cases there was no supraduodenal common duct at all (*Fig 129*) the union occurring at a point anywhere from behind the upper border of the duodenum to the part embedded in the pancreas and in 3 cases the only representative of the common duct was that part which lies in the wall of the duodenum (*Fig 130*). I did not obtain a specimen of separate entrance of the cystic and common hepatic ducts into the duodenum though this does occasionally occur.

It is usual for the cystic duct to open on the right side of the main bile-duct but in a certain proportion of cases it enters on the front, the back, or even the left side, taking a spiral course around the main duct. In 8 of my cases the junction was on the front aspect of the duct (*Fig 431*), and in 3 on the posterior. In one of the latter the union was so far around the back of the duct as to be almost on the left side (*Fig 432*).

Accessory Bile ducts.—The most interesting abnormality of the ducts is the presence of an accessory one. I have 29 examples of this. All of them are accessory right hepatic ducts. The duct leaves the liver at the extreme right end of the portal fissure, and, lying at first on a rather deeper plane than the cystic duct joins the extrahepatic ducts anywhere between the junction of the right and left hepatic ducts and the point at which the cystic duct opens into the main duct. It usually has the same relation to the right hepatic artery as the normal ducts—that is, the artery passes behind the duct.

There is no reference in the anatomy books to an accessory duct such as I found. It is true they speak of a junction of the right and left hepatic ducts at varying levels, but in all my cases this junction was at or near the normal level, and the ducts I am describing were additional. Eisendrath¹ gives drawings of these accessory ducts, but I gather from the text that they were not taken from his dissections. I have seen no reference to these ducts in the surgical literature, with the possible exception of a case of Kelm's in which he speaks of wounding the right hepatic duct where it was making a low junction with the left hepatic. It is possible this was an accessory duct as described by me mistaken by him for the right hepatic duct.

I have classified these accessory ducts into three types, according to the level at which they enter the main duct. This is done from a surgical rather than an anatomical standpoint.

1 The junction occurs in the upper half of the common hepatic duct or in the right hepatic duct (*Figs 433 and 434*). There are 9 of these. In this type the union is so high up that the duct is unlikely to be of surgical importance.

2 The junction occurs in the lower half of the common hepatic duct (*Fig 435*). There are 9 cases in this class also. The union is so near that of the cystic and common hepatic ducts as to be definitely in the field of a cholecystectomy operation.

3 The junction is at the union of cystic and common hepatic ducts (*Figs 415, 436, 437*). There are 10 of these cases. The junction is usually in the actual angle of the cystic and common ducts, but may be in the extreme lower end of the cystic duct, or in the extreme lower end of the common hepatic duct. In any case it is difficult to see how the duct could be avoided whilst clamping the cystic duct unless its presence had been previously detected.

In one specimen (*Fig 438*) the accessory duct leaves the right hepatic duct and enters the cystic duct, and of course must be cut during cholecystectomy.

The size of these accessory ducts varies. The smallest is only large enough to admit a good sized bristle. The largest is as big as the right hepatic duct. The commonest size is about half way between these limits, that is to say, it is quite a considerable duct.

There is one other abnormality of the ducts in this series, which is a curiosity rather than one of practical interest, and that is a congenital obliteration of the ducts. No

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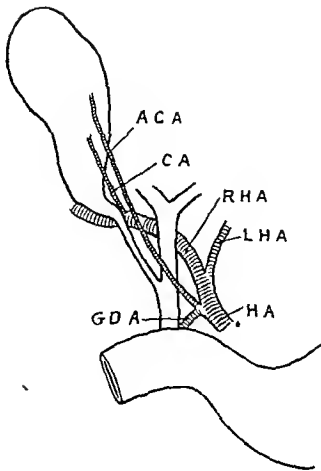


FIG 422

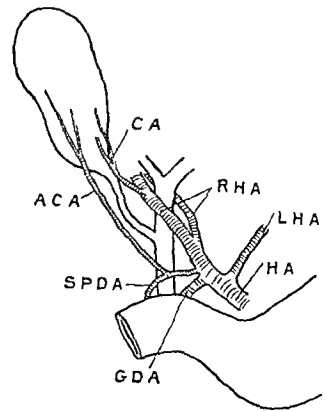


FIG 423

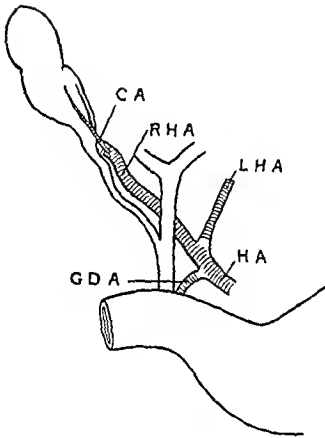


FIG 424

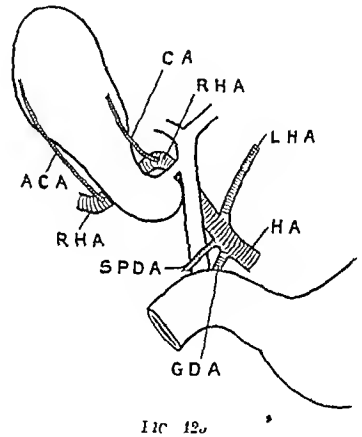


FIG 425

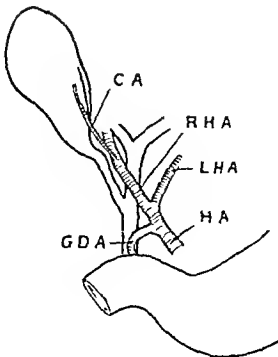


FIG 426

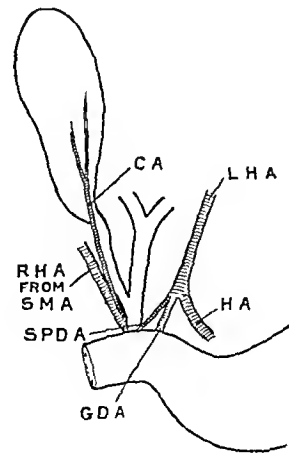


FIG 427

(CA) Celiac artery (GDA) Gastroduodenal artery (HA) Hepatic artery (LHA) Left hepatic artery (RHA) Right hepatic artery (SMA) Superior mesenteric artery (ACA) Accessory cystic artery (PA) Portal vein (CAA) Celiac axis artery (GA) Gastric artery (SA) Splenic artery (RHD) Right hepatic duct (SPDA) Superior pancreaticoduodenal artery (CD) Cystic duct

bile ducts or definite gall-bladder could be found. The infant lived thirteen weeks, having had jaundice from birth, and having passed only clay-coloured stools. At the post-mortem examination the liver was found to be fibrosed (*Fig. 139*)

THE SURGICAL SIGNIFICANCE OF THESE ABNORMALITIES

Practically all the accidents to the ducts and vessels occur during the operation of cholecystectomy with or without choledochotomy, and since cholecystectomy has almost entirely displaced cholecystostomy it is obviously the duty of every surgeon to make himself familiar with both the normal and abnormal anatomy of these parts.

It is customary, for several reasons, to begin the removal of the gall-bladder at the cystic duct, and it is here that all the traps lie. There is only one way to avoid catastrophes—that is to fix the neck of the gall-bladder with a clamp, and after snicking through the gastrohepatic omentum near this point, gently to wipe the fatty tissue towards the common duct. The cystic duct and artery now come clearly into view and can be separately secured. If there should be an accessory artery or duct it will be exposed to view before being divided and the junction of the cystic duct with the main duct can be seen distinctly. There are a few cases however in which everything is so deformed, thickened and contracted by inflammation that it is not possible to obtain a really clear definition, it is in this class of case that the most expert surgeons have probably all had unhappy experiences.

Secondary operations on gall-bladder cases have to be performed much too often, for I venture to say that 99 out of 100 could be avoided by a proper definition of the parts at the original operation combined with a better knowledge of the pathology of the gall bladder. The commonest blunders committed at the primary operation are: (1) Leaving the gall-bladder, through an inadequate appreciation of the earlier signs of cholecystitis, (2) Overlooking a stone in the cystic or common duct, (3) Injury to the common, or common hepatic, duct, (4) Injury to an accessory hepatic duct.

1 The first group is beyond the scope of this paper.

2 In the second group, difficulty might arise through the spinal arrangement of the cystic duct referred to above. A stone in that part of the duct which lies behind or in front of the common duct might be missed, or to expose it the common duct might be opened unnecessarily. Anyone unacquainted with this anomaly would be much perplexed on finding that he had opened two ducts to get at a stone which he had expected to find in the common duct. A stone in the normally disposed cystic duct or in the common duct should never be overlooked after a proper exposure of the parts.

I have already alluded to the abnormal arteries which may be encountered in opening the common duct. The superior pancreaticoduodenal and gastroduodenal arteries may be wounded in the transduodenal method of opening the common duct, for either vessel may lie in front of the duct just above the ampulla of Vater.

3 The injuries inflicted on the common hepatic or common duct practically always occur for one reason, that of not seeing clearly the various structures before applying a clamp. It is astonishing the number of surgeons who habitually neglect this simple precaution. Should the structures not be clearly defined, there are four causes to which injury to these ducts may be due: (1) A large sigmoid curve of the gall-bladder may lie closely attached to the upper part of the common duct, and on pulling up the gall-bladder the duct comes with it and may be taken for the cystic duct. In this case the common duct will be cut right across. (2) On pulling up the gall-bladder where there is a very short cystic duct, a V shaped segment of the main ducts will be drawn up as well, when possibly as much as 1 in. of the common hepatic and common ducts may be removed. (3) A clamp may be applied rather beyond the cystic duct so that a bite is taken from the side of the main duct. (4) The forceps on the cystic artery may pull off, and hasty efforts to secure it again may mean injury to the common hepatic duct through grasping the whole or a part of it with the vessel. More especially is this likely to occur when the artery arises from the right hepatic behind the duct, as it does in a small number of cases, for under these circumstances the vessel retracts out of sight.

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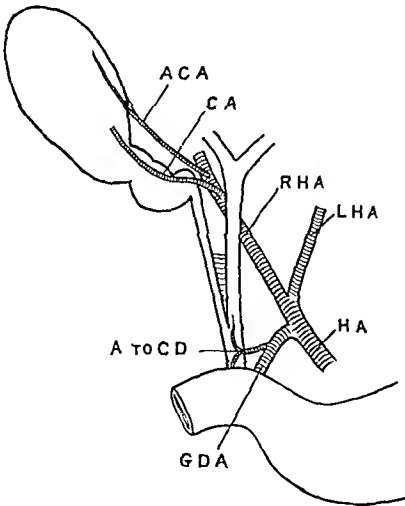


FIG 428

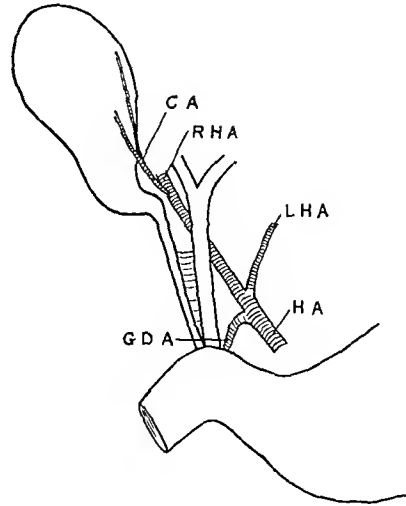


FIG 429

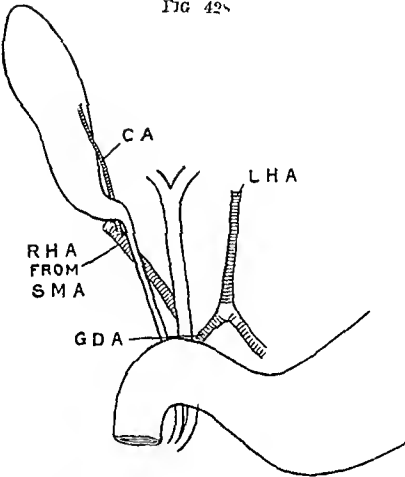


FIG 430

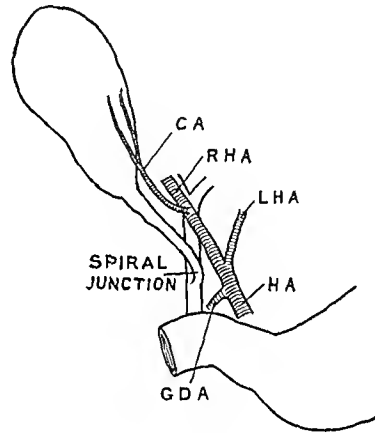


FIG 431

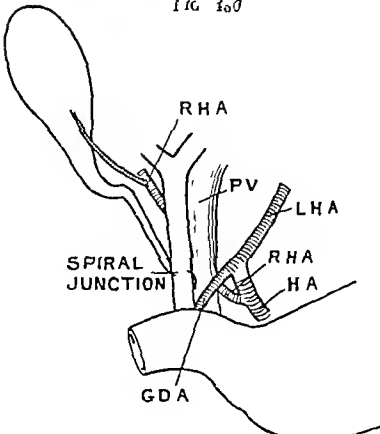


FIG 432

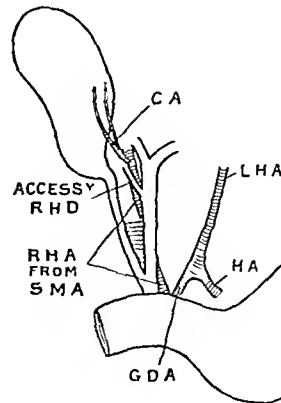


FIG 433

(C A) Celiac artery (G D A) Gastroduodenal artery (H A) Hepatic artery (L H A) Left hepatic artery (R H A) Right hepatic artery (S M A) Superior mesenteric artery (A C A) Accessory celiac artery (P V) Portal vein (C A A) Celiac axis artery (G A) Gastric artery (S A) Splenic artery (R H D) Right hepatic duct (S P D A) Superior pancreaticoduodenal artery (C D) Celiac duct

All these injuries may be successfully repaired at the time, if recognized. But they may not be revealed, for the very reason which was responsible for the catastrophe, or being repaired, a stricture may follow. Another operation will then be required if the patient survive, and anyone who has undertaken this type of operation knows how exceedingly difficult it may be.

4 In the fourth group the accessory ducts are injured. There is no literature on this subject that I know of, for I do not think surgeons as a whole are aware that these ducts exist, and certainly they are not in the habit of looking for them at operation. I have already mentioned the one possible exception of Kehr's.

I believe, for reasons which I will give presently, that these ducts are injured is often as the common duct, if not more frequently.

Since I have interested myself in this subject I have seen in accessory duct three times at operation during identification of the structures in the region of the cystic duct preparatory to clamping it. Before this I had two or three times seen the open end of what appeared to be a duct without being able to determine what it was, the end was ligatured and the patients recovered normally. Sir Berkeley Moynihan tells me he has had a similar experience. In one of my cases I saw bile coming from the cut duct and was much perturbed as I felt sure I had divided the common hepatic duct, but investigation showed this structure to be intact.

In order to strengthen my contention of the importance of these ducts to the surgeon I have been through the post-mortem records of the cases dying after cholecystectomy at the Leeds General Infirmary during the years 1908 to 1922 inclusive. During this period there were eight deaths due to bile leaking into the peritoneal cavity in considerable quantities. They were all cases of cholecystectomy without an accompanying choledochotomy. One of these deaths, which occurred in 1917, is very instructive, giving the evidence for which I was looking, and proving conclusively the importance of determining whether or no an accessory bile-duct is present. This is the post-mortem report: "On opening the abdomen a considerable quantity (one pint) of bile is found in the flank, pelvis, and around the liver. The gall-bladder has been removed. The ligature on the cystic duct is intact. Close to the liver (but not in connection with the hepatic ducts proper) is seen an open bile duct, and, on squeezing, bile exudes from it. There is nothing else of note."

In the other seven cases an extravasation of a considerable quantity of bile was noted, but the source was not determined. I think it is highly probable that some, if not all, of these cases had a divided accessory duct as in the case reported. It is more than probable that the source would be overlooked at post-mortem, for the presence of these ducts is not generally known, and therefore would not be specially sought for.

After cholecystectomy with drainage a small percentage of patients discharge bile from the wound, though the cystic duct has been ligatured. This begins at once or within a few hours of the operation, and is obviously bile from a duct. It has been attributed to bile from the raw surface of the gall-bladder bed, but it is too profuse for that, and, moreover, is not intimately mixed with blood as it should be if from this source. The other explanation given is that the ligature has slipped off the cystic duct. This has never seemed to me an adequate explanation, for the cystic duct is easy to tie, and there is only a very low pressure behind the ligature. In view of what has been said above, a much more reasonable supposition is that of an injured accessory duct.

Though it is bad surgery to injure these ducts, the probability is that little harm would result in the majority of cases, provided a drainage tube were left in. Many surgeons have advocated from time to time closure of the abdomen after cholecystectomy. Probably the fashion is more prevalent now than ever before. I suspect these surgeons are not aware of the possibility of the presence of accessory ducts, as the subject has never been raised in this connection, and closure of the abdomen with one of these ducts cut would be a disaster. Such an accident would in all probability be explained as a leak from the cystic duct.

To this the argument might be advanced by those who favour closure that bile would

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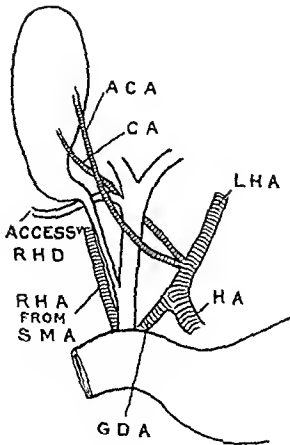


FIG 131

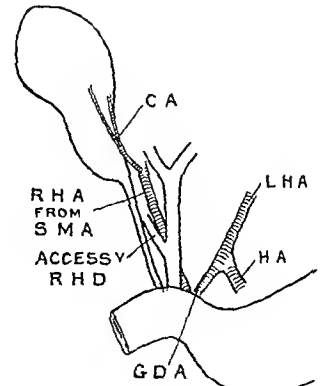


FIG 132

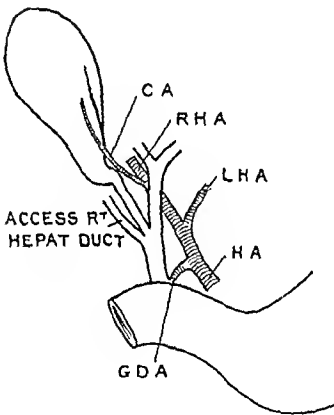


FIG 133

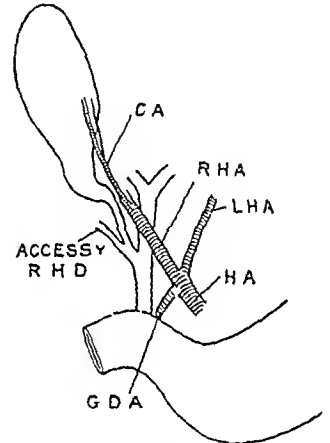


FIG 134

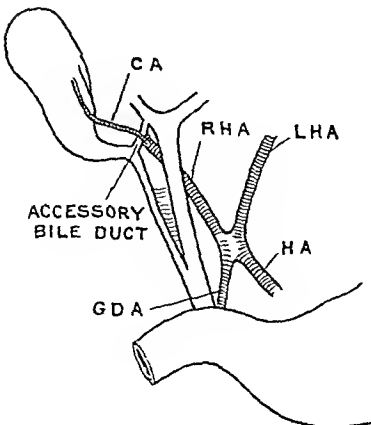


FIG 135

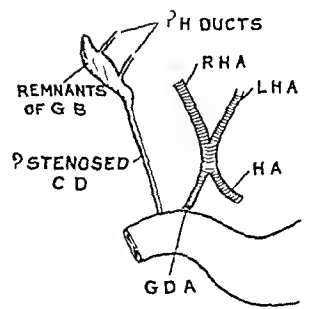


FIG 136

(CA) Cystic artery (GDA) Gallbladder artery (HA) Hepatic artery (LHA) Left hepatic artery (RHA) Right hepatic artery (SMA) Superior mesenteric artery (ACA) Accessory cystic artery (PV) Portal vein (CAA) Celiac axis artery (GA) Celiac artery (SA) Splenic artery (RHD) Right hepatic duct (SPDA) Superior pancreaticoduodenal artery (CD) Cystic duct

be seen to flow from the open end of the duct during the operation and that the application of a ligature would make it safe to complete the abdominal closure. But this does not necessarily follow, for it is well known that after putting a tube in the common duct not a single drop of bile may appear sometimes for twenty-four or thirty-eight hours. Though this is not so likely to happen after cholecystectomy alone, I have no doubt that in those cases associated with much hepatitis the secretion of bile is more or less temporarily suspended. Moreover there is no objection of any moment to leaving a piece of dental rubber as a drain for one or two days through an incision which displaces the rectus outwards. I have never seen a haemorrhage and it will save a life now and again.

Two interesting questions arise to which no definite answer can be given as yet —

1 What happens to that part of the liver drained by one of these ducts after a ligature has been applied?

2 What becomes of the duct if it be divided and left open?

As to the first question, only a limited part of the liver drains into this duct, and one would expect the healthy organ would be able to compensate in its other parts, for the capacity of the liver cells to multiply in accordance with necessity is equalled by no other organ in the body. I performed an experiment with the object of finding out whether bile cut off in one part of the liver could make its way round to another. I ligatured the common duct low down, and also the cystic and left hepatic ducts. I then injected methylene blue into the upper part of the common duct. The only way into the liver was up the right hepatic duct, and yet bile appeared in both the right and left lobes. I have not had time to follow up this investigation and as I did not know the exact pressure at which the dye was injected, too much importance must not be attached to it. The subject is however, worth further investigation, for it is known that bile can make its way from one group of liver-cells to another in the immediate neighbourhood.

Suppose that one of these accessory ducts were ligatured when the liver was not healthy, as, for instance, in a case of prolonged obstruction to the common duct by a stone. In such a case temporary suppression of bile is known to be not uncommon after operation, and I think it is quite possible that extra work thrown suddenly on the damaged liver, as would be the case after ligaturing a fairly large accessory duct, might cause a total suppression.

As to the second question, probably the open duct gradually closes as a result of the cicatricial changes in the operated area, and the bile may subsequently find its way round to other parts of the liver, or possibly this part of the liver undergoes cirrhotic changes.

Though quite a large number of injuries to the bile ducts have been collected and reported by various writers, notably Eisendrath and Elliott, these probably only represent a small fraction of the total number of injuries that have been inflicted, for surgeons are ashamed of committing such errors, and rightly so, and do not feel disposed to advertise them. So long as surgeons continue to clamp and ligature structures in this region *en masse*, catastrophes are certain to occur. It is bad artistry, and can only be avoided by seeing everything, and being familiar with all the abnormalities.

I wish to express my thanks to Dr M. J. Stewart, Professor of Pathology at the Leeds University, for his courtesy in assisting me to obtain the specimens for dissection.

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SOME OBSERVATIONS ON THE
TREATMENT OF ACUTE APPENDICITIS

By R J McNEILL LOVE, LONDON

THE object of this paper is to endeavour to elucidate two points in particular with regard to acute appendicitis (I) *The best time to operate*, (II) *The best methods of drainage* when such is required. In order to acquire sufficient data from which to draw reliable deductions, I have reviewed the cases admitted to the London Hospital between 1919 and 1922—a total of 1503.

I THE BEST TIME FOR OPERATION

All surgeons agree that within the first twenty-four hours of the onset of acute appendicitis, the sooner the appendix is removed the better. In the majority of these cases hyperæsthesia of a band of skin above Poupart's ligament can be obtained, indicating that the peritoneum surrounding the appendix is stretched by œdema or distention of that organ. This hyperæsthesia indicates that the inflammation is limited to the appendix, and removal of that organ will abruptly terminate possible developments. Operation at this stage usually allows complete closure of the wound without drainage, and is relatively safe, as shown in Table 1 —

Table 1 —CASES OPERATED ON WITHIN 24 HOURS OF ONSET

NO OF CASES	CLOSED WITHOUT DRAINAGE	LOCAL DRAINAGE	PFELVIS DRAINED	FATAL COMPLICATIONS	MORTALITY	STAY IN HOSPITAL
176	121	38	17	Secondary abscess and general peritonitis 1	0.57 per cent	16.4 days

As treatment of cases seen within the first twenty-four hours is not a subject of controversy I shall not allude to them, and further figures only refer to cases of longer standing. However, I would suggest that the presence of hyperæsthesia is a safer guide to the condition of the appendix than arbitrary length of time, e.g., twenty-four hours.

With regard to these later cases, it has been the custom of recent years to operate as soon as the diagnosis of appendicitis was reasonably assured, with a view to removing the offending organ, preventing further infection, and draining inflammatory exudates. The mortality of operating at once is somewhere about 5 per cent. Grey Turner,¹ in a series of 681 cases, found it to be 5.13 per cent, 1000 cases collected by Hugh Lett² at the London Hospital in 1912 showed a mortality of 4.3 per cent. However, these series include cases of only twenty-four hours' duration, and hence the mortality of later cases must be somewhat higher than the figures quoted. In the present series of cases operated on immediately (excluding those in Table 1), the mortality was 6.24 per cent.

It is becoming more recognized that the peritoneum is willing and able to deal with a considerable amount of inflammatory exudates, and therefore many surgeons now delay operation and carefully watch developments. This allows the acute inflammatory reaction to subside in which case appendicectomy is performed after an interval of about a week.

Although in these more enlightened days of surgery, expectant treatment of a surgical condition does not appeal to surgical instincts, yet it may be remembered that, before

surgical interference became the rule, patients treated on these expectant lines usually recovered. In 1890 Sahli collected 6710 cases of perityphlitis which were treated expectantly, the mortality being 591, or only 8.8 per cent, and McBunney³ states that 99 per cent of cases of perityphlitis are due to inflammation of the appendix. These figures are all the more striking because the importance of Fowler's position and the dietetic restrictions was not then appreciated, and purgatives, etc., were commonly misused.

When patients are given the benefit of delay the following routine is rigidly adhered to. The patient is placed in Fowler's position, water only is allowed by the mouth, and fomentations may be applied to relieve pain. After twenty-four hours, if the signs and symptoms of infection are abating, this treatment is continued until the temperature and pulse are normal, the patient is allowed fluid diet (milk, Benger's food, etc.) when he expresses a wish for it, which time usually coincides with the approach of temperature to normal. Operation is performed one week later unless physical signs still suggest an abscess, which usually absorbs within another week. As Edmund Owen⁴ pointed out, one must consider both general and local signs of infection. If, after a period of delay the temperature and pulse remain elevated, or an abscess increases in size, then operation should be performed.

In 228 cases in which operation was delayed, 151 (66.8 per cent) responded to expectant treatment. The majority of the 77 who failed to respond were doubtless subjected to energetic treatment before arriving at hospital, which told against their settling down after admission. This suggests the criticism that even if some cases are tided over until infection subsides, the remainder who must be operated on during acute infection will show a higher mortality due to the delay, which may more than counterbalance the advantage gained in the case of the former. However, statistics fail to bear this out, and, even in cases where an operation was performed of necessity at the end of twenty-four hours or later, the mortality is approximately the same as in cases operated upon arrival in hospital. The twenty-four hours' delay, under appropriate treatment as outlined above, seems to be compensated for by the fact that it allows the patient to regain his mental calmness and settle down after his journey to the hospital, which may be sufficiently tedious to exhaust a patient who is acutely ill.

It is interesting to note that all statistics show that operation on the third day carries a much higher mortality than either earlier or later cases. Owen⁴ quotes 708 cases which were operated on the third day, with a mortality of 10.7 per cent, in this series of cases the mortality was 9.8 per cent. This may be due to the fact that the resistance of the patient is at its lowest ebb at this period, and that manipulations during operation flood the tissues with toxins before the formation of an adequate quantity of antibodies.

The following tables compare the results of delayed operation with those performed as an emergency, i.e., as soon after arrival at hospital as expedient.

Table 2—CASES IN WHICH OPERATION WAS SUCCESSFULLY DELAYED

NO. OF CASES	DAYS IN HOSPITAL BEFORE OPERATION	DRAINAGE	INCIDENTAL COMPLICATIONS	FATAL COMPLICATIONS	MORTALITY	STAY IN HOSPITAL
151	8.6	Local 54 Pelvic 7 Total 61	Intestinal obstruction 2 Fecal fistula 1 Secondary abscess 1 Phlebitis 2 Total 6	General peritonitis 2 Pulmonary embolism 1 Total 3	1.9 per cent	25.1 days
		= 40.4 %				
			= 3.3 %			

5 When the wound can be closed, the patient is spared the discomfort of removal of tubes repeated dressings etc with a corresponding economy in hospital expenditure

6 Length of stay in hospital is considerably diminished, and the patient is discharged with a healed wound instead of, as frequently happens, a granulating sore Hence the period of convalescence is shortened

One minor disadvantage of the delayed treatment is that the patient after being successfully tided over the acute infected stage, feels restored to health and refuses the subsequent operation This happened in one case and fifteen months later the patient returned with a fatal recurrence Appendicectomy must of course always be insisted on and it is estimated that 80 per cent of cases of acute appendicitis relapse within two years unless the appendix has been removed *

It would, of course, be a dangerous policy to suggest to the general practitioner and public that cases of appendicitis are not in urgent need of hospital treatment This is undeniable, but the treatment should be expectant rather than operative Cases should still be sent to hospital at the earliest possible time preferably while still exhibiting the band of hyperæsthesia indicative of an intact appendix If this is lost, the patient should be treated accordingly, and the progress closely watched so that operation can be performed at any period if necessary Attention was drawn to the clinical significance of this cutaneous hyperæsthesia as long ago as 1903 by James Sherren,⁶ and two years later the same surgeon advocated expectant treatment as described above *

II THE BEST METHODS OF DRAINAGE

In considering this all-important question it is necessary to bear in mind the four principles involved in efficient drainage (1) This should be as dependent as possible (2) It should be obtained by the most direct route, (3) The risk of secondary hemorrhage must be borne in mind, (4) The abdominal wall should be weakened as little as possible

The site requiring drainage naturally depends upon the area infected, and this brings one to a short consideration of the various methods of approach to the appendix Ignoring those cases in which the appendix lies on the left side, or in the region of the gall-bladder, three methods of approach may be employed—Battle's, the right rectus, and the gridiron

Battle's incision, in which the sheath of the rectus is incised vertically and the muscle displaced inwards undoubtedly gives a good exposure of the appendix area, and is useful in cases of uncertain diagnosis especially in females, as it allows ready exploration of the pelvis However, it appears to present the following disadvantages (1) In cases where the infection is localized to the right iliac fossa the site is reached through the general peritoneal cavity, which is thus liable to contamination, and if drainage is made through this incision the tube passes through a zone of previously healthy peritoneum, hence there is risk of infecting the general peritoneal cavity (2) When drainage is obtained through this incision the tube lies in the vicinity of the deep epigastric vessels, and secondary hemorrhage has occurred from this cause (3) There is risk of injury to the intercostal nerves, especially the lowest of the series, causing paralysis of the rectus muscle and lower part of the abdominal wall, predisposing to subsequent inguinal hernia

The right rectus incision consists of a vertical incision over the lower part of the rectus muscle, the sheath is divided, and the muscle, with its nerve supply, displaced outwards This gives ample exposure for the removal of the appendix, the chronic appendix associated with gastric ulcer is frequently removed through this incision, i.e. a continuation downwards of the upper abdominal incision Conversely, in the case of erroneous diagnosis when the appendix has been examined and the pathological lesion is in the upper abdomen this area can be reached by enlarging the incision upwards This incision, however, when employed in acute cases of appendicitis, again has the disadvantage of being placed too near the mid-line, and therefore, in many cases, the area of infection is reached through healthy peritoneum

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Table 7—DRAINAGE THROUGH RIGHT RECTUS (INCLUDING BARTH'S) INCISION

NO OF CASES	INCIDENTAL COMPLICATIONS		TOTAL COMPLICATIONS		MORTALITY	STAY IN HOSPITAL
109	Faecal fistula		General peritonitis		6.4 per cent	28.9 days
	Secondary abscess	3	Secondary abscess	3		
	Intestinal obstruction	3	Ileus	2		
	Phlebitis	1	Subdiaphragmatic abscess	1		
	Pleural effusion	1		1		
		Total 9		Total 7		
	= 8.2 per cent					

Table 8—CLOSURE OF PRIMARY WOUND AND SUPRAPUBIC DRAINAGE

NO OF CASES	INCIDENTAL COMPLICATIONS		TOTAL COMPLICATIONS		MORTALITY	STAY IN HOSPITAL
303	Intestinal obstruction	2	General peritonitis	9	5.3 per cent	26.5 days
	Secondary abscess	3	Ileus	2		
	Faecal fistula	3	Secondary abscess	2		
	Phlebitis	2	Intestinal obstruction	1		
	Subdiaphragmatic abscess	1	Subdiaphragmatic abscess	1		
		Total 11	Doubtful	1		
	= 3.6 per cent			Total 16		

The most striking feature of the above figures is the far larger number of complications in Tables 6 and 7, than in Table 8. Three of the most important complications may briefly be considered.

1 *Mechanical Intestinal Obstruction*—Combining Tables 6 and 7, we find that this occurred in 37 per cent of cases, compared with 0.9 per cent in the last group. The passage of the drainage tube through the peritoneal cavity undoubtedly generates adhesions, hence the shorter the drainage tube the less the likelihood of their formation. It is found that the distance from the usual gridiron incision to the pouch of Douglas is two to three inches longer than by the suprapubic route, and the length of drainage tube through a right rectus incision is longer in proportion to the exact position and length of the incision. The routes are demonstrated in the accompanying illustration (Fig. 440). A second factor in the promotion of adhesions is that a tube in contact with parietal peritoneum is less likely to cause adhesions than one passing through coils of intestine. If infection is retrocecal, or additional drainage of the right iliac fossa is indicated, this can be obtained by a stab wound in the loin.

2 *Faecal Fistula*—This is four times as common in Table 6 as in Tables 7 and 8 combined. Faecal fistula may be due to sloughing of part of the cecum, inability to bury the stump satisfactorily, pressure of the tube, all of which may be associated with distention of the cecum due to temporary paralysis of the gut. Certain of these causes

are unavoidable whatever drainage is attempted, but it would appear that pressure of the tube or the maintenance of infection along the track of the tube in the neighbourhood of the cæcum strongly predisposes to the formation of a fistula.

The majority of fistulae close spontaneously within a fortnight, and in only one of these series of cases was an operation necessary for closure. However, a faecal fistula means delayed healing and increased infection around a wound, and hence greater risk of post operative hernia.

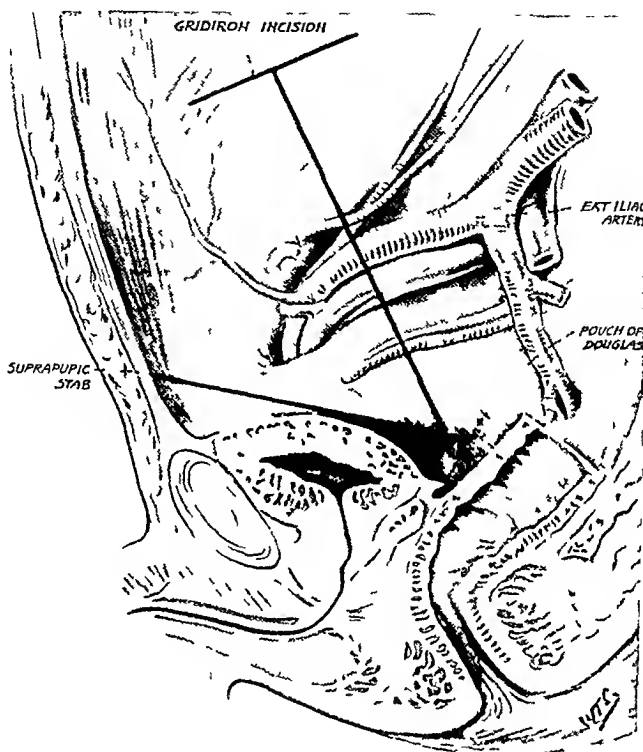


FIG 440.—Diagram illustrating the shorter method of approach to the pouch of Douglas by the suprapubic route compared with drainage through a gridiron incision, also proximity of the tube through the latter incision to the external iliac vessels. As this is an antero-posterior view the line indicating the gridiron route of drainage is foreshortened and hence the actual distance is longer than that represented by the line.

3 Secondary Haemorrhage—The risk of secondary haemorrhage from the external iliac vessels consequent on a drainage tube crossing the pelvic brim is well known⁹ (see Fig 440), and in Table 6 one such fatal case occurred. The external iliac artery was ligatured but the patient died eight hours later. Secondary haemorrhage has also been reported from the external iliac vein¹⁰. This danger of secondary haemorrhage is enhanced in females, whose broader pelvis increases the obliquity of the tube, and in whom the route from the pelvis to the gridiron incision is even longer and more devious than in the male.

Some surgeons obviate the risk of secondary haemorrhage by early removal of the tube, but it seems difficult to foretell how long it may be necessary to retain a tube, and a premature removal predisposes to a secondary abscess.

Drainage through the right rectus incision allows the tube to enter the pelvis without dangerous proximity to these vessels but secondary haemorrhage has followed drainage through this incision from the deep epigastric vessels¹¹.

With suprapubic drainage the tube lies in proximity to no large vessels, and hence the risk of secondary haemorrhage is negligible. A further feature of suprapubic drainage

is that the original incision is closed, which favours its primary union, and this factor, combined with diminished complications, accounts for the shorter stay in hospital. Suppuration of the wound inevitably leads to weakening of the abdominal walls, with increased risk of post-operative hernia. Scudder and Goodall,¹² in reviewing the after-results of 640 cases, found that incisional hernia occurred in 17 per cent of cases in which the drainage tube passed through the original wound.

B LOCALIZED ABSCESSSES

These are naturally usually located in the right iliac fossa, and therefore the gridiron incision gives direct approach. No definite surface markings are taken for the incision, but this should be made to the outer side of the swelling. In all cases where pus is suspected it is wise to smear the superficial tissues with BIPP or a similar substance in order to diminish the risk of a superficial abscess occurring on the wounds breaking down. When the abscess is opened care should be taken to ascertain that no pus has trickled over the brim of the pelvis where it may be overlooked. Hence the importance of rectal examination prior to operation may be emphasized, as the collection of pus may then be recognized. Deaver¹³ estimates that in 2 to 5 per cent of cases the appendix is so buried that a prolonged search for it is unjustified, but much depends upon the skill and experience of the surgeon.

Table 9—DRAINAGE OF LOCALIZED INFECTION THROUGH WOUND

NO OF CASES	INCIDENTAL COMPLICATIONS	TOTAL COMPLICATIONS	MORTALITY	STAY IN HOSPITAL
249	Rectal fistula 14 Secondary abscess 7 Intestinal obstruction 3 Phlebitis 2 Subdiaphragmatic pleurisy 1 Total 27 = 10.8 per cent	Secondary abscess 3 Intestinal obstruction 2 General peritonitis 2 Ileus 1 Pyelophlebitis 1 Total 9	3.6 per cent	24.1 days

Table 10—DRAINAGE OF LOCAL INFECTION THROUGH STAB WOUND IN LOIN

NO OF CASES	INCIDENTAL COMPLICATIONS	TOTAL COMPLICATIONS	MORTALITY	STAY IN HOSPITAL
27	Rectal fistula (through gridiron incision) 1 Phlebitis 1 Total 2 = 7.8 per cent	Nil	Nil	20.9 days

With regard to the question of drainage of an abscess localized in the right iliac fossa, a stab wound in the loin gives the shortest and most dependent route, especially when the appendix is retrocaecal. The stab wound can readily be made on the points of a pair of

sinus forceps introduced through the wound, care being taken not to injure a prolapsed kidney if such a condition co-exists. The advantages of drainage through a stab wound in the flank are suggested by the figures in *Table 10*, although unfortunately the number of cases is rather small, their shorter stay in hospital is noteworthy.

SUMMARY

1 All cases of acute appendicitis should be operated on immediately if a brand of hyperæsthesia is still present.

2 Under appropriate treatment the majority of remaining cases subside, and the appendix may be removed seven to ten days after the temperature and pulse become normal. As compared with emergency operation this line of treatment shows a lower mortality, fewer complications, and a shorter stay in hospital.

3 In cases in which expectant treatment fails, twenty-four hours' delay does not appear to influence the prognosis adversely.

4 Except in cases where difficulty is anticipated (e.g., recurrent cases), or where the diagnosis is doubtful, the gridiron is the incision of choice, as manipulations can then be limited to the infected area. In other cases a right rectus incision, displacing the muscle outwards, is preferable to Battle's, there being less risk of injury to the nerves.

5 Stab drainage, suprapubic or in the flank, favours primary union of the original wound, thus diminishing the length of stay in hospital and risk of subsequent incisional hernia. Also adhesions and faecal fistulae are less likely to develop, and the risk of secondary hæmorrhage is obviated.

I am indebted to the various surgeons at the London Hospital for permission to include in these series cases which were under their observation and treatment.

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A NOTE ON THE OPERATION FOR THE RADICAL CURE OF FEMORAL HERNIA.

By ERNEST W. HEY GROVES, BRISTOL

ABOUT fifteen years ago, when I had to operate upon a strangulated femoral hernia in a stout woman, I divided Poupart's ligament in order to obtain a better view of the neck of the sac and its contents. It was a case in which the viability of the gut was in serious question, I feared that a piece of bowel higher than I had access to from the groin might be damaged, and I could not pull down further intestine without fear of rupture. Therefore I had recourse to the expedient of dividing Poupart's ligament with Gimbernat's ligament close to their attachment to the pubic spine. This gave an exposure so excellent that full investigation was possible, and after dealing with the sac and its contents, I was able to attach the conjoined tendon to Cooper's ligament with much greater facility than if this had had to be done under the overhanging tendon of the external oblique.

It occurred to me on the occasion of this operation that, quite apart from dealing with strangulated bowel, this procedure afforded greater precision in performing a radical cure than any other operation I had hitherto seen or performed.

I have therefore carried out this femoro-inguinal operation as a routine ever since. I think it is probable that many other surgeons have done the same thing, but I have read no description of this operation, whilst visitors who have watched it have been interested in it as a new procedure.

I therefore venture to describe it in detail and to give the results in a consecutive series of cases.

The hernia is exposed by a vertical incision about four inches long, the middle of which corresponds with the fold of the groin. A very little dissection then reveals the hernial sac, lying on the pectineus muscle, with Poupart's ligament crossing its neck, and on its outer side the femoral vein joined by the long saphenous vein (*Fig 441*).

The attachment of Poupart's ligament to the pubic spine is then cut close to the bone and the ligament turned outwards (*Fig 442*). This gives a clear view of the neck of the sac, which is then opened and its contents dealt with according to their condition. Should resection of the bowel be necessary, this can be done perfectly well by the exposure thus afforded. When the sac has been cut off and its neck ligatured, the femoral ring and external can be obliterated (*Fig 443*). The conjoined tendon is defined and sewn down to Cooper's ligament and the upper edge of the pectineal fascia. The outer one or two stitches also take up the femoral sheath on the inner side of the femoral vein so as completely to close the abdominal cavity from the thigh. Poupart's ligament is then laid over the line of suture between the conjoined tendon and Cooper's ligament, beginning from the vein and working inwards towards the spine of the pubis (*Fig 444*). As the inner attachment of Poupart's ligament has been divided, there is no tension in this structure and it can be made to follow the line of the pubic bone, and the final attachment of its cut end will be about $\frac{1}{2}$ in further outwards than it was originally. In my later cases, instead of cutting Poupart's ligament, I have chipped off a small part of the pubic spine (*Fig 445*), and at the final stage of the operation the detached piece of bone is fixed by means of a single ivory nail.

The advantages which this combined femoral and inguinal operation afford would seem to me to be as follows —

1 In cases of strangulated hernia it gives ample room to deal with damaged bowel and, if necessary, to do a resection.

2 It gives all the advantages of the inguinal approach, i.e., the possibility of closing the femoral canal from above, without the necessity of trying to drag up the hernial sac through the femoral canal

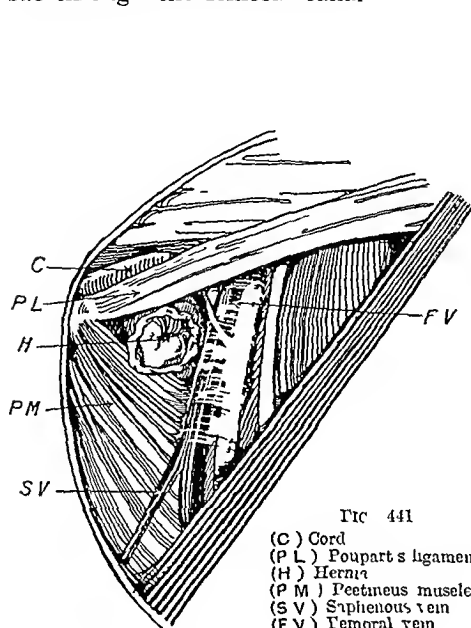


FIG 441

(C) Cord
(PL) Poupart's ligament
(H) Hernia
(PM) Pectineus muscle
(SV) Saphenous vein
(FV) Femoral vein

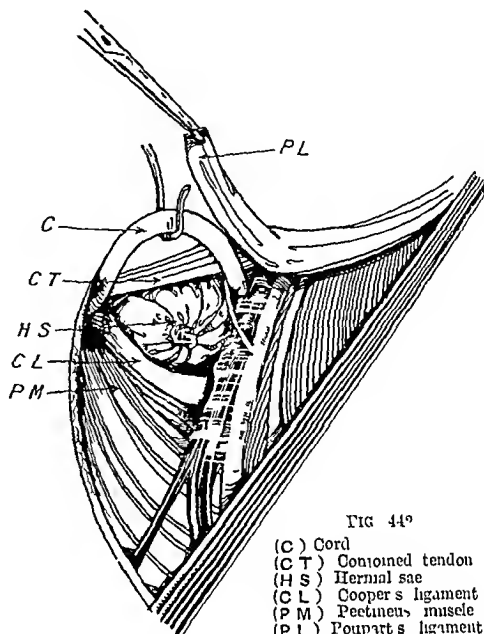


FIG 442

(C) Cord
(CT) Conjoined tendon
(HS) Hernial sac
(CL) Cooper's ligament
(PM) Pectineus muscle
(PL) Poupart's ligament

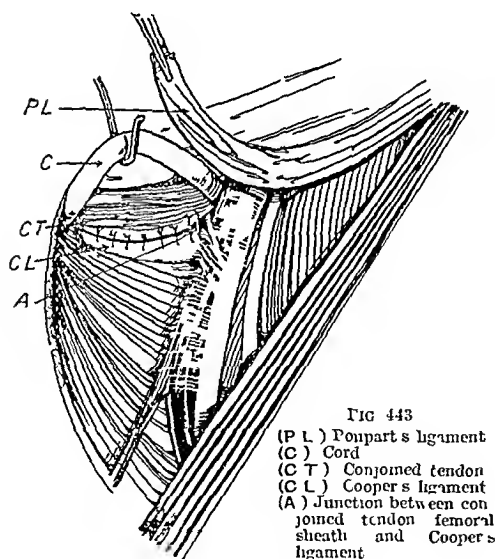


FIG 443

(PL) Poupart's ligament
(C) Cord
(CT) Conjoined tendon
(CL) Cooper's ligament
(A) Junction between conjoined tendon femoral sheath and Cooper's ligament

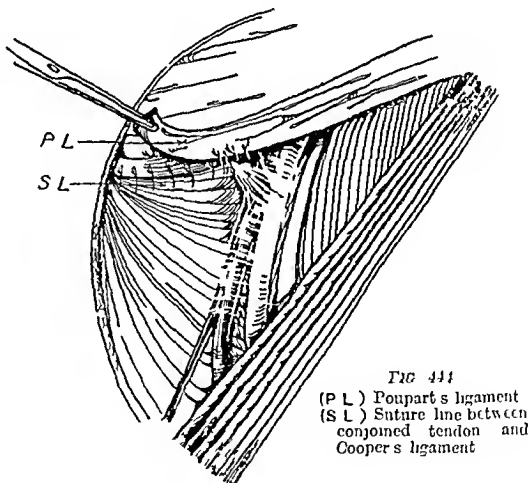


FIG 444

(PL) Poupart's ligament
(SL) Suture line between conjoined tendon and Cooper's ligament

3 It allows the suture of the conjoint tendon to Cooper's ligament to be undertaken with great precision, unhindered by the overlying Poupart's ligament

4 Poupart's ligament, being freed from tension, can be snugly sutured as an extra covering over the line of suture between the conjoint tendon and Cooper's ligament

Results of the Operation—I have looked up all cases operated upon by myself during the period 1912 to 1922, and I have been able to see and examine 22. Thus

is of course a small number, but sufficient to give some criterion of the permanency of cure

In one case only was the complete radical operation not attempted, this being on account of the gangrenous condition of the large omental contents. It was intended to complete the radical operation at a later date, but the patient did not care to have this further operation, and she still has a hernia about the size of a duck's egg

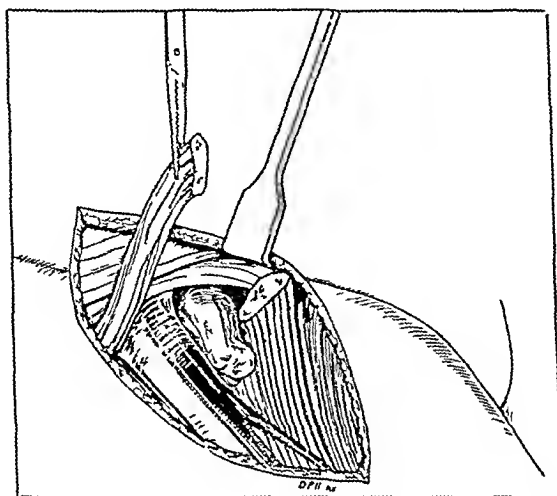


FIG 445 —Right femoral hernia removal of a piece of bone from the pubic spine

All the other 21 cases have remained soundly healed and free from recurrence. The sex incidence was 21 women and 1 man. The ages varied between 23 and 73. In 9 cases the operation was necessitated by strangulation.

All the cases have been hospital patients, and they have, with one exception, been able to return to housework or work in factories after the operation. The exception is a woman, now 72 years old, who is bedridden with heart disease.

In 3 cases the patients have borne children since the operation, without any recurrence of the hernia or inconvenience from the site of the operation.

ABNORMALITIES OF FIXATION OF THE ASCENDING COLON THE RELATION OF SYMPTOMS TO ANATOMICAL FINDINGS

By ADAMS A McCONNELL AND T GARRATT HARDMAN, DUBLIN

SUMMARY

- 1—THE NORMAL ASCENDING COLON
 - a* Anatomy
 - b* Radioscopic examination
- 2—DEVELOPMENT AND FIXATION
- 3—VARIATIONS IN FIXATION AND RESULTING TYPES OF ASCENDING COLON
- 4—VARIATIONS IN FORM OF ASCENDING COLON, CLINICAL PICTURE
- 5—TRACTION OF ASCENDING COLON ON OTHER STRUCTURES
 - a* Superior mesenteric artery
 - b* Duodenum
 - c* Gall bladder
 - d* Pyloric end of stomach
 - e* Right kidney
- 6—REMARKS ON RADIOSCOPIC TECHNIQUE
- 7—GENERAL OBSERVATIONS
- 8—GENERAL TREATMENT

STIMULATED by Waugh's paper on the mobile ascending colon, and realizing the possible significance of his conception in abdominal surgery, we decided to observe the ascending colon in every abdominal case, and to inquire whether its anatomical condition was in any way responsible for the symptoms or disease encountered. The result of our investigation can be presented most readily by describing what we consider the normal ascending colon, the manner of its development, the variations of the normal which we have encountered, and the symptoms associated with these variations.

1 THE NORMAL ASCENDING COLON

a **Anatomy**—Anatomists describe this portion of the gut as it is seen in the dead subject, surgeons visualize it as it appears during a laparotomy, both see it when the subject is recumbent. Radioscopy alone reveals its position in the erect living subject.

The normal ascending colon lies in direct contact with the posterior abdominal wall, and is held in position by the reflexion of the parietal peritoneum. The hepatic flexure and the beginning of the transverse colon are similarly fixed to the anterior surface of the right kidney and to the second stage of the duodenum respectively, by means of this fixation the ascending colon is maintained in a vertical position as a practically straight tube presenting some slight curves or flexures (*Fig. 416*). Such is the conception which is obtained from observation in the cadaver.

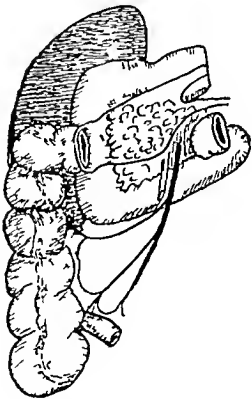


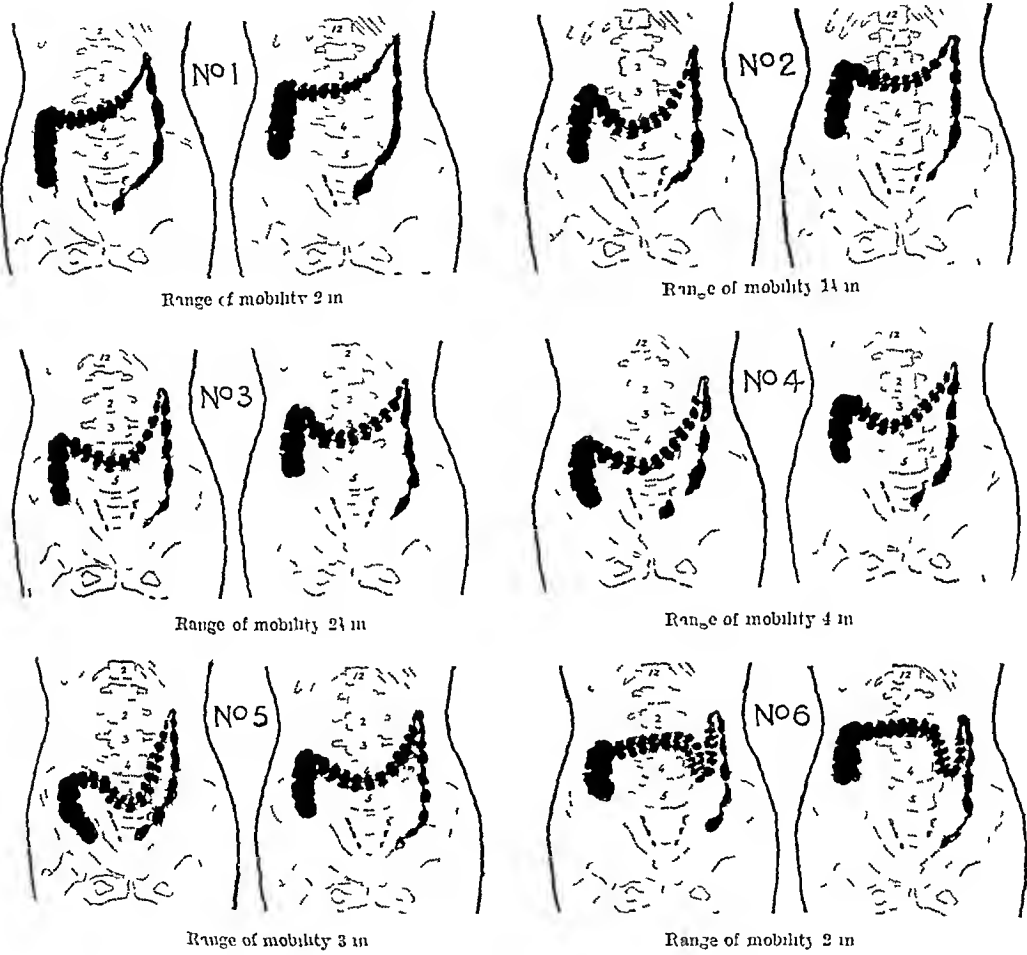
FIG. 416
Normal ascending colon

b **Radioscopic Examination**—This was carried out with the fluorescent screen in a series of individuals who presented no symptoms of abdominal or other disease. The results are as follows —

Males	Ages between 21 and 38 (Fig 447) —		
	Average length, including caecum	erect	7 inches
	position of hepatic flexure above iliac crest,	erect	1½ "
	" " " " " "	recumbent	4 "
	range of vertical excursion	" "	2½ "

FIG 447—MALE COLONS Nos 1 to 6

(In each instance the diagrams on the left show patient in the erect position those on the right in the recumbent position)



The ascending colon formed practically a right angle with the transverse colon. The former was practically straight, and the diameter of the caecum was but slightly greater than that of the succeeding segment of gut. There was very little lateral mobility of the ascending colon on palpation.

Females	Ages between 20 and 29 (Figs 448, 449) —		
	Average length, including caecum,	erect	7 inches
	" " " " " "	recumbent	8 "
	(the longest was 9 in the shortest 6½ in —recumbent)		
	Average position of hepatic flexure above iliac crest,	erect	¾ "
	" " " " " "	recumbent	3 "
	range of vertical excursion	" "	2½ "

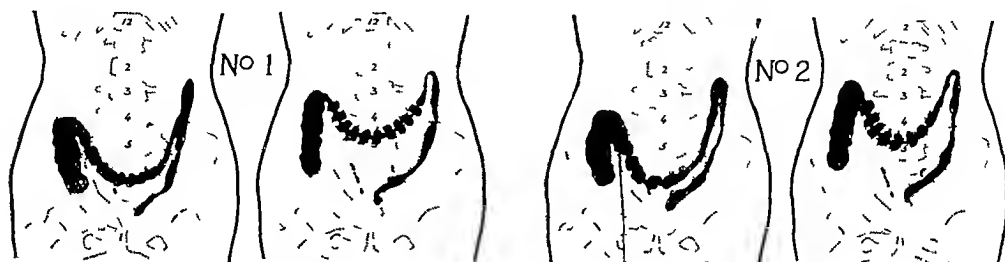
In 82 per cent the ascending colon was straight. In 18 per cent it presented one or more flexures along its course. The angle between the ascending and transverse portions

of the colon was less than a right angle. The diameter of the cæcum was practically the same as that of the ascending colon. There was but slight lateral mobility on palpation in 92 per cent.

In determining the length of the ascending colon by radioscopy one must be careful, in making the measurement, that the summit of the barium shadow actually coincides

FIG 448—FEMALE COLONS Nos 1 to 6

(In each instance the diagrams on the left show patient in the erect position those on the right in the recumbent position)



Range of mobility 2 in

Angulated type Range of mobility 3 in



Range of mobility 2 in

Range of mobility 3 1/2 in



Collapsed type Range of mobility 3 in

Range of mobility 2 in

with the top of the hepatic flexure. This portion of the gut, if empty, will (it is hardly necessary to point out) cast no shadow, and hence the summit of the opaque content might be erroneously taken as the top of the hepatic flexure, but if there is an accumulation of gas in the flexure the bowel will be clearly outlined and there is no danger of such an error. We have found that the best time in which to make measurements is twenty-four hours after the ingestion of the meal, when, as a rule, the transverse colon as well as the cæco-ascendens is rendered visible by its opaque contents. These measurements we regard to be of the first importance in ascertaining the degree of fixation or lack of fixation of the hepatic flexure. In pronounced cases of what we may refer to as *mobile colon*, the position of the flexure varies enormously with the posture of the patient, or, in other words, flexion occurs at two different points in the colon, according as the patient is erect or recumbent. From our series of normal individuals we have conceived—whether rightly or wrongly—that the average normal ascending colon is practically constant in length in

2 DEVELOPMENT AND FIXATION OF THE ASCENDING COLON

About the time of birth the cæcum is situated in front of the right kidney, near the gall-bladder, and the proximal part of the colon possesses a complete mesentery. Then both the cæcum and ascending colon gradually descend towards the right iliac fossa. Adhesions form between the parietal peritoneum and the posterior aspect of the ascending colon and its mesentery. These two adherent peritoneal surfaces disappear, so that the colic vessels and the colon itself become retroperitoneal, and the normal adult condition is reached. An ascending mesocolon is as abnormal as a cleft palate.

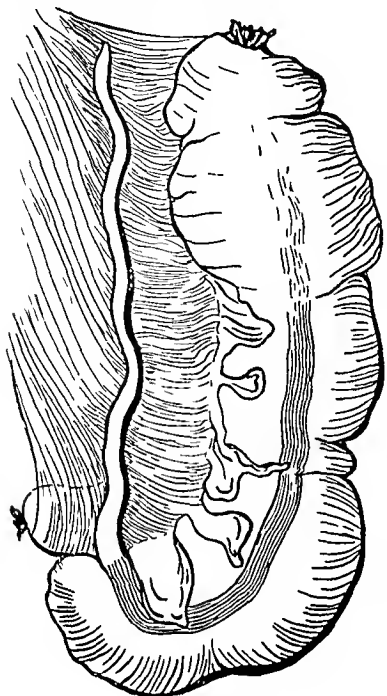


FIG 450—Primitive mesentery entire

3 VARIATIONS IN FIXATION AND RESULTING TYPES

The variations of this normal process which we have met surgically are—

a The new adhesions may persist in excess to the lateral aspect of the ascending colon and constitute varying degrees of parietocolic folds. We have observed these folds so frequently in newborn children



FIG 451—Prolapsed type

and infants under two years in the anatomical departments of Trinity College, Dublin, and the Royal College of Surgeons, that we regard them as congenital.

b The new adhesions may not form at all, or may remain too attenuated to function, so that the primitive mesentery persists in its entirety (Fig 450), when one of two things may happen. (i) The whole ascending colon sinks to the limit permitted by its mesentery, and remains straight—the *prolapsed* type (Fig 451). This type is usually associated with lax abdominal muscles. (ii) The cæcum may be prevented from sinking, and the hepatic flexure approximates to the cæcum, so that the ascending colon resembles a collapsed concertina—the *collapsed* type (Fig 452), or it becomes sharply bent at one point—the *angulated* type (Fig 453).

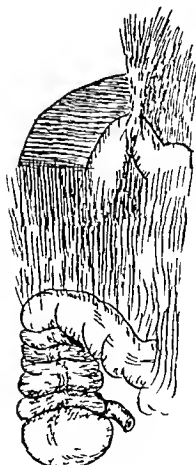


FIG 452—Collapsed type

c The new adhesions may fix only the lower or middle portion of the ascending colon, and the upper part may remain free, so that the hepatic flexure tends to fall down



FIG 453—Angulated type of ascending colon.

in front of or to the side of the lower portion, and produce a sharp angulation of the gut—the *angulated* type. Thus this type may or may not have an attachment to the plicates

X ray Appearances—Radioscopic examination with the fluorescent screen shows that the prolapsed type of ascending colon remains practically straight both in the erect and recumbent positions (*Fig 449, No 12, and Figs 454 and 455*) Its length is very little



FIG 441—Twenty four hours after meal —Erect



FIG 442—The same case —Recumbent

altered The vertical excursion of the hepatic flexure may be as much as 6 in Its distinctive feature is its lateral mobility—one can push it to the middle line or beyond, sometimes it might be termed a 'floating' colon, so pronounced is its mobility in every direction The collapsed and angulated types appear normal with the patient recumbent when the erect position is assumed however, the appearance of both is that of an irregular mass in the right iliac fossa (*Figs 456, 457, 458*) Palpation under the screen reveals the difference in the two types (*Fig 449, Nos 2 and 5, and Fig 459*) We have encountered the prolapsed type in 58 per cent, the collapsed in 3 per cent, and the angulated in 31 per cent whilst 8 per cent were apparently normal on x ray examination



FIG 443—Twenty four hours after meal —Erect

The type of ascending colon present is therefore a function of its fixation modified in some degree by the tonicity or atony of the musculature of the anterior abdominal wall When the ascending colon has no attachment to the posterior abdominal wall it is supported altogether by its attachments to the right kidney and second stage of the duodenum, by its own mesentery and through the proximal part of the transverse colon, by the gastrocolic omentum and pyloric end of the stomach The results of faulty fixation fall therefore into two general classes, namely (1) *Variation in the actual form of the ascending colon*—the angulated and

collapsed types, and (2) *Excessive traction on the structures to which the ascending colon is attached*, varying inversely with the degree of attachment to the posterior abdominal wall and with the tonicity of the abdominal musculature

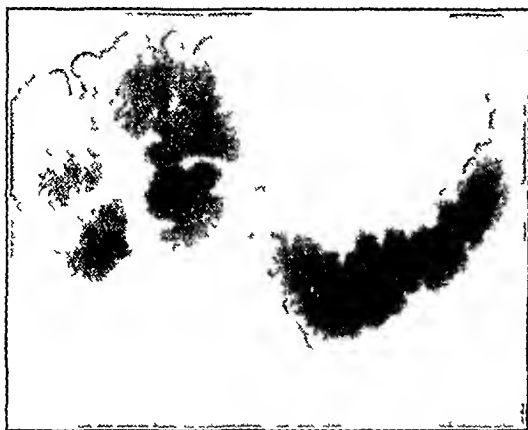


FIG 406 —Forty eight hours after meal — *Ingulated type, erect*



FIG 406A —The same case —*Recumbent*

4 VARIATIONS IN THE FORM OF THE ASCENDING COLON

Mass Movement in a Normally Fixed Colon—The contents of the colon are semi solid, and are propelled onwards, not by short peristaltic waves occurring frequently, but by very infrequent vigorous contractions which appear to start in the cecum or proximal end of the ascending colon, and travel over a considerable distance, driving the contents



FIG 407 —Forty eight hours after meal —*Erect*



FIG 407A —The same case —*Recumbent*

of the bowel before them in one mass or column. At the moment when the contractions start, a remarkable change occurs in the transverse colon. The normal haustration disappears, and the bowel, which a moment before had formed a sagging loop between two fixed points, appears to shorten and to become like a rigid tube straight across the abdomen. This shortening of the transverse colon is due presumably to contraction of

the longitudinal muscular bands. The whole contents move rapidly for a distance of perhaps 6 to 12 in., and the movement stops as suddenly as it commenced, the haustral contractions reappearing, and the colon returning to its normal position and quiescence.



FIG 488 —Erect



FIG 489 —The same case —Recumbent

These movements are only very rarely seen, and have been observed by one of us on not more than three occasions in the course of hundreds of examinations. The result of this contraction is that, no matter how acutely angulated the hepatic flexure may be, it assumes a right-angled bend during contraction.



FIG 490 —Erect

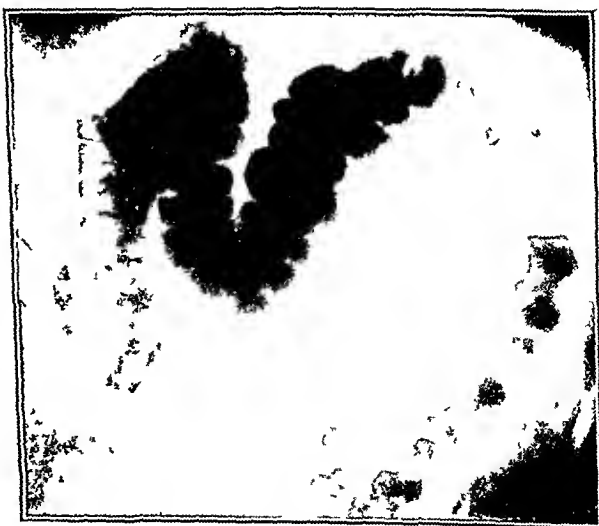


FIG 491 —The same case —Recumbent

Mass Movement in the Imperfectly Fixed Colon—We have never been fortunate enough to observe this movement in the deformed or mobile type of ascending colon, it seems probable however that, when the hepatic flexure is not fixed both the ascending and the transverse segments lose a point of purchase and complete mass movement does

not occur, but a small portion of the colonic contents passes along the gut at each attempt. The presence of a strong parietocolic fold halfway up the ascending colon may afford a fixed point, and the mass movement takes place, but probably not so perfectly as when the hepatic flexure itself is fixed.

It is in cases of the collapsed and angulated types of ascending colon that most real distention is observed. In these the contents of the cæcum have to be forced past a kink or kinks and against the dead weight of the superimposed colon. In the absence of a point of fixation, contraction of the colonic musculature does not obliterate these kinks but exaggerates them so that they constitute a true obstruction resulting in dilatation of the cæcum and the proximal part of the ascending colon.

When the individual assumes the recumbent posture the hepatic flexure falls into the normal position, the kink or kinks disappear, and the obstruction is relieved. This periodic relief by recumbency may account in part for the long period that often elapses before these congenital defects produce clinical evidence of their presence.

The Relation of Symptoms to the Angulated and Collapsed Ascending Colon—

Case 1—Female, age 30. About six months before admission she began to suffer from loss of appetite, and discomfort in the right iliac fossa after food. All kinds of food disagreed with her, and her diet became restricted to small quantities of tea and milk. After a few weeks the discomfort became actual pain, it came on during breakfast and immediately after other meals, and was accompanied by nausea. After she had taken a few mouthfuls at breakfast she felt so sick that she could not eat any more. In the course of an hour or two she resumed the interrupted meal. When she stayed in bed for breakfast she experienced neither pain nor nausea, and had less discomfort after other meals. If she remained in bed all the time she could take food freely. Never vomited. Was never constipated until these symptoms appeared. Since their onset she became thinner and somewhat anæmic. Tenderness was present in the right iliac fossa over a distended cæcum. Radioscopy demonstrated the angulated type of ascending colon in the erect posture. It straightened out when the patient lay down. Excursion of hepatic flexure 4 in. A large amount of the meal remained in the proximal colon after thirty-six hours. The rest of the gastrointestinal tract appeared normal.

Operation—Upper two-thirds of ascending colon possessed complete mesentery. Parietocolic fold fixed lower one-third. Stomach and duodenum normal. Appendix normal. Appendicectomy—colopexy. Result after one year and nine months—quite well.

When the symptoms were referred to the right iliac fossa, the ascending colon was of the angulated or collapsed type. The ascending colon usually has its heaviest load in the morning. When the patient gets up, the angulation or collapse becomes marked, when he takes his breakfast contraction of the colon begins and he suffers from colicky pains in the right iliac fossa or the lower part of the abdomen. If dilatation of the cæcum has occurred, the patient experiences a sensation of sinking or fullness, accompanied by nausea and dry eructations. If he lies down the pain is relieved, if he has breakfast while lying down the pain is prevented, hence the statement "I cannot do without my early cup of tea." Colonic contraction stimulated by ingestion of food empties the ascending colon before the patient assumes the erect posture and therefore before angulation or collapse of the gut takes place. If he goes to stool after breakfast in bed, he has a free and satisfactory motion. If he has got up for breakfast, the evacuation is but of the contents of the lower bowel and he suffers from discomfort in the right iliac fossa until the bowels move again. This morning discomfort may be repeated, or will ultimately be repeated, after every meal. If the normal evacuation of the bowels takes place after the evening meal, it is sometimes during that meal that pain has its onset or exaggeration. Once these symptoms appear, they usually continue duly until the patient learns how to control them. Sometimes, however, he suffers from attacks at irregular intervals.

These symptoms have no marked relation to constipation, often indeed the patient will not admit that he is constipated. Radioscopy, however, frequently demonstrates some degree of stasis in the proximal colon, the more pronounced the stasis, the more does the patient suffer from general symptoms of toxæmia—persistent headache, nausea, loss of appetite, lassitude and wasting. As time goes on he becomes anæmic and nervous.

There is a definite relation in some cases between physical or mental exhaustion and the onset or recurrence of these attacks. Some of the patients developed symptoms only

when tired. Apparently colonic tone was sufficient to overcome whatever colonic obstruction was present, until fatigue removed the former and left the latter unchanged. The typical symptoms, in brief, are those of subacute obstruction in the ascending colon—discomfort, pain, or a sinking sensation in the right iliac fossa coming on during or immediately after a meal, especially breakfast, and *relieved by lying down*. Nausea is common, vomiting unusual. Sometimes adhesions have fixed the colon so kinked, and the symptoms are independent of posture. Physical examination reveals distention of the cæcum. A pericæcal fold was found in nearly every case of the angulated type operated on. Jackson's veil-like membrane was present in 15 per cent of all cases. Not every patient with an angulated or collapsed colon has these symptoms, but every patient with these symptoms has an angulated or collapsed colon.

The fact that this clinical picture was always associated with either the angulated or collapsed type of ascending colon, and not with the prolapsed straight type, has convinced us that it is only when an obstruction can be demonstrated in this segment that marked symptoms are referred to the right iliac fossa. So long as the ascending colon is straight, its mobility, though interfering with complete evacuation, does not markedly prevent it.

The complete clinical picture is preceded by less definite and suggestive symptoms. When about twenty years of age the patient becomes gradually conscious of vague discomfort in the lower part of the abdomen—a feeling of heaviness or slight nausea during or immediately after a meal. He finds that he has to go to stool twice instead of once. Ultimately he discovers that relief comes if he lies down. Palpation reveals slight tenderness in the right iliac fossa, the distended cæcum is noted, but often ignored. Medicine does no good, and then a normal appendix is removed, which operation may mark the beginning of a surgical pilgrimage. Other patients are perfectly well for twenty or more years, when the condition of the colon is discovered in seeking for the cause of sudden acute pain in the right iliac fossa. It is always safer to make a diagnosis of acute appendicitis in doubtful cases, but if the appendix be not diseased, the culpability of the colon should be suspected. In such acute cases we have been impressed with the apparent well-being of most of the patients. Although they may writhe with pain, the temperature is not raised, the rate of pulse is not quickened, and their appearance is not toxic.

Symptoms somewhat similar to those given as typical have been ascribed to a 'distended cæcum' apparently idiopathic, and to incompetency of the ileocæcal valve. We have not once observed the latter in these cases, but the former is constant. J. C. Roux, in a paper reviewed by Robert Hutchison in the *Medical Annual* of 1922, gives an account of what he has termed cæcal constipation. He notes the occurrence of some of the above symptoms and the relation of discomfort in the right iliac fossa to posture. Among the causes of this variety of constipation he includes a mobile ascending colon, the presence of false membranes, inflammatory pericæcolitis, and cæcal ectasis. All the cases in our series which had cæcal ectasis had the angulated or collapsed types of mobile colon and demonstrable obstruction. Surely dilatation of a tube makes one suspect obstruction.

Patients with the prolapsed type of ascending colon seldom referred their symptoms to the right iliac fossa. When they did, however, the clinical picture was never so complete as in the angulated type. Vague discomfort, a sensation of heaviness, sinking, or pain after food, sometimes relieved by recumbency, marked these cases. None of them presented acute symptoms.

Eighteen patients referred all or some of their symptoms to the right iliac fossa. Of these, one had the collapsed type of colon, twelve the angulated, and five the prolapsed. Four of the cases with the angulated type were admitted as acute appendicitis. At operation no lesion was found in the appendix. Gastro-enterostomy for gastric ulcer had been performed in two cases more than a year before, with relief of epigastric symptoms, but those referred to the right iliac fossa persisted or became prominent. Two patients had had the appendix removed without any relief. Apart from the eighteen there were six cases in which the ascending colon was angulated and no symptoms were referred to the right iliac fossa.

Treatment—Many patients have learned by experience how to relieve the symptoms, and many physicians are empirically successful. The administration of liquid paraffin, a dose of salts before breakfast in bed, and recumbency for half an hour after each meal may keep a patient free from discomfort, and some there be who can adopt such a regime. Removal of a normal appendix does not affect the progress of the case. Cæcophication by itself is based on a false idea of the cause of the dilatation. We do not compress the head for hydrocephalus, nor plicate a dilated ureter. Cæcophication may be useful along with colopexy. Fixation of the cæcum alone leaves the condition unchanged when the colon is angulated or collapsed, whilst in the prolapsed type angulation or collapse is produced, an empty stocking will not stand in a shoe. Resection of the cæcum and ascending colon is unwarrantable in early cases, one does not amputate the foot because it is flat. When, however, long-standing obstruction has left the cæcum simply an inert atonic sac, an ileo-cæcal resection is rational. Division of parietocolic folds turns an angulated colon into a prolapsed one, and substitutes one set of symptoms for another. The whole ascending colon must be made straight and then fixed to the posterior abdominal wall. It must be remembered that the diagnosis of the angulated and collapsed types of ascending colon cannot always be made at operation, for then the colon may have fallen into a normal position, and one can say no more than that it is mobile. As a rule deformity is revealed only by *x*-ray examination.

More than a year has elapsed since the operation in twelve of these cases. Eleven of the twelve report 'well'. Colopexy was performed in each. The twelfth case had, in addition to mobility of the colon, a well-marked Lane's kink and ileal stasis. The ileal band was divided and the appendix removed, the colon was not fixed. This patient's symptoms returned within ten days of leaving hospital.

5 TRACTION ON THE STRUCTURES TO WHICH THE ASCENDING COLON IS ATTACHED

When the ascending colon fails to acquire a wide surface of apposition to the unyielding posterior abdominal wall, its suspension depends partly on its own mesentery, and on its peritoneal attachments to the renal fascia and second stage of the duodenum. Normally the proximal portion of the transverse colon is largely supported by the more fixed hepatic flexure. When the latter is mobile, it ceases to be a support, and becomes partly dependent on the beginning of the transverse colon, which, in turn, drags on the gastrocolic omentum and on the pyloric portion of the stomach. This strain on the stomach exerts itself primarily on the greater curvature, which, however, can yield to it, being essentially a mobile portion of the stomach, but the lesser curvature of the stomach is relatively fixed by the attachment of the lesser omentum, and consequently is subject to more tension.

In a considerable number of cases there are congenital peritoneal folds passing from the gall-bladder to the duodenum or to the hepatic flexure through these the mobile ascending colon may drag indirectly or directly on the gall-bladder.

The fact that a mobile ascending colon does exert traction on the structures to which it is attached was established by noting the position of the hepatic flexure and of the ileocecal junction under the radioscopic screen with the patient erect. At operation the hepatic flexure was placed in its predetermined position, and the effect of this replacement on the duodenum and pyloric end of the stomach observed. The ileocecal junction was similarly replaced in order to note the effect on the superior mesenteric artery and third stage of the duodenum.

If the ascending colon be in its normal relation to the posterior abdominal wall, its position is affected little, if at all, by laxity of the anterior abdominal wall. Radioscopy and laparotomy have frequently shown a fixed colon in cases with chronic laxity of the anterior abdominal wall. Whatever be the condition of the anterior abdominal wall, the presence of a mesentery allows the ascending colon to sink to an extent not possible to the relatively fixed structures to which it is attached, and to exert a certain degree of traction on them. Strong abdominal muscles may keep the ascending colon up, but they do not keep it up all the time, nor can they keep it straight.

The effects of traction in general reach their maximum under the following conditions: (i) When there is a complete ascending mesocolon, (ii) When the abdominal wall is lax; (iii) When the colon is heavy with retained contents; (iv) When the patient is in the erect posture.

The structures which have been affected by traction of the mobile ascending colon in our series of cases have been: (a) The superior mesenteric artery, causing obstruction to the third stage of the duodenum; (b) The second stage of the duodenum; (c) The gall-bladder; (d) The pyloric end of the stomach; (e) The right kidney.

a. TRACTION ON THE SUPERIOR MESENTERIC ARTERY (CHRONIC GASTROENTERITIS, ILLER CHRONIC DUODENAL ILLER (WHICH) ARTERIAL DUODENAL ILLER)

Though obstruction of the third stage of the duodenum by the superior mesenteric artery has been recognized since 1849, when Rohdendorf described it as a possible cause of acute dilatation of the stomach, Bloodgood, in 1907, was the first to demonstrate its association with a mobile ascending colon. In September, 1921, one of us published 4 cases of this association, to which we now add another 15. Of the total number of cases in the series, 19 showed a pronounced degree of duodenal dilatation caused by the superior mesenteric artery.

The blood vessels of the normal ascending colon lie behind the parietal peritoneum, as does that segment of the gut. When a complete primitive mesentery is present the vessels pass between its leaves and share in that degree of tension to which the mesentery is subject. The superior mesenteric artery lies opposite the 1st lumbar vertebra and crosses the third stage of the duodenum opposite the 2nd. The lumbar segment of the spine is convex forward, and the summit of its convexity corresponds to the anterior part of the body of the 2nd lumbar vertebra. The superior mesenteric artery, therefore, has to pass forwards as well as downwards from its origin, and, after crossing the duodenum, incline somewhat backwards.

(Fig 460.) Any tension

on the artery must therefore produce obstruction of the duodenum. In the normal case no such tension exists. The mesentery of the small intestine is long enough to allow the greater part of the duodenum to lie on the pelvic floor, and the jejunum rests on the duodenum. If the mesentery were not long enough, tension on the vessel would be inevitable. In one case the cecum and several feet of the duodenum occupied the site of a large inguinal hernia, and obstruction to the duodenum by the artery was demonstrated. When the ascending colon is mobile, however, its mesentery, and particularly the ileocolic artery, is not always long enough to follow the excursion of the gut without tension, and hence the tension on the ileocolic artery is communicated to the superior mesenteric trunk, and obstruction to the third stage of the duodenum results.

The greater the anterior convexity of the lumbar segment of the spine, the more likely is duodenal obstruction to occur, and when this does take place, acute flexion of the thorax on the abdomen will diminish it by abolishing the anterior lumbar convexity. The more



FIG 460. To show the relation of the superior mesenteric artery to the duodenum and to the lumbar spine. The defect here leads to the pyloric form.



FIG 461. To show the relation of the third stage of the duodenum to the superior mesenteric artery.

the cæcum inclines to the back of the pelvis the more tension is there on the superior mesenteric artery. The superior mesenteric vein is never the obstructive agent, we have repeatedly seen it passing over the dilated portion of the duodenum while the artery lay in a deep groove. This vein passes directly upwards to the portal at this point, and does not incline at all backwards—its course is at a tangent to the anterior surface of the third stage of the duodenum. Occasionally the vein and the artery are separated by a distance of an inch or more as they cross the duodenum.

Fifteen of the cases of arterial duodenal ileus seen by us have had the prolapsed type of colon. Four had the angulated type. We have but once encountered a case of arterial duodenal ileus in which the terminal ileum and cæcum did not lie in the pelvis as observed under the radioscopic screen. Parietocolic folds sometimes prevented the upper part of the ascending colon from being drawn to the middle line, and the recumbent position of the patient on the operating table sometimes brought the ileocecal junction up, so that the colon at operation did not seem unduly mobile or prolapsed. Radioscopy occasionally reveals more than laparotomy.

The whole duodenum down to the artery was dilated so that the supramesocolic portion often presented in the wound and could even be withdrawn through it (Fig 461). The duodenum from its pyloric end to the crossing of the superior mesenteric artery seemed to have undergone a bucket-handle rotation.

Relation of Symptoms to Anatomical Findings—

Case 2—Male, age 28. Sturdy and healthy in appearance. For six years before admission, suffered from attacks of abdominal pain at irregular intervals. Each attack was characterized by the sudden onset of violent pain in the right side of the epigastrium, coming on about a quarter of an hour after food, and accompanied by vomiting, which only slightly relieved it. The interval between attacks was sometimes a few weeks, occasionally several months. Each attack came on when he was constipated, although he was not of a constipated habit, and lasted for about four days. It often started at night when he was asleep, and forced him to assume a sitting posture. He had an attack in hospital, and there was no question of the extreme severity of the pain. During the attack there was tenderness over the painful area, but no muscular rigidity. The right kidney was movable, but not markedly so, and no urinary symptoms were present. The anterior abdominal wall was of excellent tone.

ANAL REPORT—Normal shaped, tonic stomach. Rapid evacuation of contents at first, but small residua retained six hours after ingestion of meal. Marked tenderness localized over pylorus and first stage of duodenum. No deformity. Ascending colon prolapsed but straight. Lateral mobility present. Positive diagnosis not justified, but there is a suggestion of ulcer at pylorus or first stage of duodenum.

OPERATION—Stomach and biliary tract normal. The duodenum presented in the wound, it was mobile and dilated down to crossing of superior mesenteric artery. No sign of gastric or duodenal ulceration. Ascending colon was freely mobile, with complete primitive mesentery. A thin veil like Jackson's membrane over upper part. A long appendix running up along lateral surface of ascending mesocolon. Appendicectomy—colopexy. Appendix normal. Result after two years and two months. No return of pain or abdominal trouble of any kind, eat and do anything.

Attacks of severe pain in the epigastrium, extending into the hypochondrium on each side were common. Sometimes the pain was felt intensely in the back at the same or a higher level. *It was relieved by posture* in every case but one. In 12 out of 19 cases the method adopted by the patients to this end was to sit on a low stool or in bed, to draw the knees up to the chin and clasp the legs with the arms. These patients had found this method of relief for themselves and thus it was that drew our attention to the significance of the curve of the lumbar spine. In this position the lumbar convexity was abolished, and the pressure of the thighs probably acted as a support to the prolapsed colon. Of the 7 remaining cases 2 lay prone with a pillow or their fists under the abdomen for relief. 1 stood up and walked about. Three patients were relieved by lying down but mentioned no special position. One patient was not relieved by any posture. Pain was increased in two cases by lying on either side—in one by lying on the right and in another by lying on the left side. In but three cases was it not increased by the supine position. Fig 460 illustrates our explanation of the production or increase of pain when the patient assumes this position. The cæcum slips from the right iliac fossa

into the pelvis and tends to fall into the hollow of the sacrum, thus drawing the superior mesenteric artery more tightly across the duodenum. The pain had no relation to food in 11 cases, in 4 it came on from one to two hours after a meal, and in 1 food relieved the pain. Vomiting, copious and recurrent, was marked in 13 cases, and absent in 6. One patient had vomited nearly every day for five years. Eructations of gas were usual. All the patients complained of a sensation of distention after food, necessitating loosening of the clothing. Anything taken into the stomach when the pain was present was usually regurgitated immediately. Some of the patients were free from pain as long as the bowels moved satisfactorily. Constipation always precipitated an attack. In the majority of cases the first attack took place in childhood.

The following case shows how closely the clinical picture may resemble that of duodenal ulceration —

Case 3.—Male, age 63. Thirteen months before admission he began to experience attacks of pain at the right side of the epigastrium and back of shoulder-blades. He described the pain as that of wind, which was relieved by eructations. Pain came on two hours after a meal and was relieved by it. He was awakened by the pain between 1 and 2 a.m. He always carried an apple about with him to eat when the pain began. The pain was always worse after a meal containing meat. These attacks lasted for a few days and recurred in a few weeks. Appetite was consistently good. Relief of pain by posture was constant. *Most relief was experienced by sitting up, hugging his knees, and leaning to the left side.* Pressure of a pillow on the abdomen when lying prone also relieved him. Lying on the right side increased the pain. Since the age of 15 he had taken a small dose of Glubers salts every morning. Failure to secure a duly executed had given him abdominal discomfort when a boy. On the morning of admission to hospital he had a sudden severe pain in the epigastrium, and vomited a frothy slime. The pain was so severe that he collapsed at his work. On admission he presented the typical appearance of a perforated gastric or duodenal ulcer, the history supported the latter diagnosis. Laparotomy revealed the duodenum enormously dilated down to the superior mesenteric artery, no sign of duodenal ulceration could be seen. The whole ascending colon fell out of the wound. On the lesser curvature of the stomach a large indurated ulcer was found. An aperture was made in the transverse mesocolon and a penetrating ulcer of the posterior wall of the stomach was found, its floor being formed by the transverse mesocolon. This ulcer was infolded and a gastro-enterostomy performed proximal to it. There was no connection between the ulcer on the posterior wall and that on the lesser curvature. The patient made an excellent recovery.

The above case is presented in full because of the typical history of duodenal ulcer in the presence of arterial duodenal ileus and gastric ulcer, and in the absence of duodenal ulceration. [We are indebted to Sir Thomas Myles for permission to include this case in our series.]

Gastro-enterostomy had been performed previously in the two following cases —

The first patient was well for seven years after the operation, when he developed attacks of pain in the middle of the epigastrium radiating along the left costal margin. There was no relation between the ingestion of food and the onset of pain, but if he took any food during an attack he vomited immediately. Vomiting relieved the pain, as also did the squatting posture. Rhinoscopy demonstrated pyloric relaxations resulting in large gushes of food from the stomach, enough passing through it at a time to fill the duodenum down to the position of the superior mesenteric artery. Food also passed through the gastro-enterostomy opening. The ascending colon was prolapsed and mobile. At operation, arterial duodenal ileus was demonstrated and colopexy performed. This patient has had no further trouble since the operation fifteen months ago.

The second patient had had an anastomosis made between the jejunum and the proximal end of an hour-glass stomach two years previously. Marked amelioration followed this operation, but there was still some pain and a sensation of fullness in the epigastrium, accompanied by flatulent eructations and nausea. The pain was relieved by the squatting position. The patient was losing weight and strength and was becoming progressively anæmic. In addition to the epigastric symptoms she had attacks of severe pain in the right iliac fossa and a 'sinking sensation' immediately after food. The appendix was removed but proved to be normal. Shortly after the appendicectomy we read Wigham's paper and sent for the patient. Her condition had in no way improved. A further examination showed stasis for twenty-four hours in the distal sac of the hour-glass stomach and a mobile ascending colon of the collapsed 'concertina' type with a distended cecum. The abdomen was again opened, and the duodenum found markedly dilated down to the crossing of the superior mesenteric artery. The ascending colon had a complete primitive mesentery. Colopexy was performed. Since this operation, nearly two years ago, the patient has steadily improved and has had no return of abdominal discomfort.

The outstanding clinical features of these cases may be summarized thus: Attacks of acute epigastric pain, not related to food, culminating in vomiting and definitely relieved by flexion of the spine. We consider the squatting posture pathognomonic of this condition. Every patient who relieved his pain in this fashion had arterial duodenal ileus. There were six patients who had none of these symptoms and yet had arterial duodenal ileus. They were operated on for symptoms referred to the right iliac fossa or for chronic constipation, and the third stage of the duodenum was found dilated down to the superior mesenteric artery. Each had a mobile ascending colon. Observation of the entire duodenum in every abdominal case is necessary in order to determine what degree of dilatation of the third stage is to be considered pathological.

Treatment of Arterial Duodenal Ileus.—Relaxation of the ileocolic artery is the essence of treatment. This may be done by keeping the cæcum up by an abdominal belt, or less effectively by diminishing the contents of the cæcum, and therefore its weight, by suitable purgatives. The rational treatment seems to be to put the cæcum and the ascending colon into their normal relation to the posterior abdominal wall and fix them there. As all our cases of arterial duodenal ileus had a mobile ascending colon, we have not performed duodenojejunostomy. X-ray examination after colopexy and the disappearance of the patients' symptoms have convinced us that *this operation alone is curative in the majority of cases*. In one case vomiting, pain in the epigastrium, and a sensation of fullness persisted after colopexy. Radioscopy showed a retention of barium in the stomach after twenty-four hours, as it was before the operation. We operated six months after the first operation with the object of performing a duodenojejunostomy, and were impressed by the fact that the third stage of the duodenum had returned to its normal size. The stomach was dilated and, as had been noted at the first operation, a small scirr was present on the first stage of the duodenum. There was very slight induration, and no other sign of duodenal ulceration. It is probable, however, that a small ulcer was present. Gastro-enterostomy was performed, with excellent result.

There is no doubt that duodenojejunostomy will cure duodenal ileus. We are indebted to our colleague Sir Conway Dwyer for the opportunity of seeing the operation performed by him for this condition about eight years ago. That case has been well since. He has performed duodenojejunostomy on three other occasions with good results. If, however, prolapse of the ascending colon is the causative factor in this condition, colopexy is a simpler primary procedure.

Removal of a cause does not necessarily remove an effect, and in some cases longstanding atony of the duodenum may preclude its return to normal after the mesenteric strain is removed. Duodenojejunostomy is then indicated, as it is in cases due to shortening of the mesentery of the small intestine.² Murphy³ and Wheeler⁴ have each reported cases in which the obstructive agent was a peritoneal band to the right of the superior mesenteric artery.

Colopexy was performed in 14 of these cases: in 2 over two years ago, in 5 over a year and a half, in 4 over one year. Of recent cases 3 are included. Thirteen patients are well since the operation; they can eat what they like without discomfort. One patient died as the result of operation. She was a woman of 26 who had suffered from indigestion and constipation since childhood, during adolescence she became progressively anæmic. When admitted to hospital she was so weak that she had to be kept absolutely at rest for a month before even an x-ray examination was ventured upon. This examination demonstrated duodenal ileus. After this she was treated medically for six months, without much effect. At operation the stomach was dilated and the duodenum down to the superior mesenteric artery was greatly dilated. Colopexy was performed, and she died, apparently of shock, thirty-six hours afterwards. The post-mortem examination revealed no hemorrhage or other abdominal catastrophe.

Of the 13 cases now well, one developed adhesions between the small intestine and the scirr of the abdominal wound and was re-operated on twice in a year for obstructive symptoms. The last operation was performed fourteen months ago. The ascending colon was found definitely fixed in a normal position. Another patient, otherwise well, complains

of stiffness and weakness in the right side a year after operation. Of the remaining 5 cases, one, a girl, age 12, was admitted as acute intussusception. Laparotomy revealed duodenal ileus and a mobile ascending colon loaded with hard faeces, the colon was not fixed, as the child's condition was poor. Colonic lavage relieved the symptoms. Avoidance of constipation has kept this patient well for over two years. The same treatment was adopted in another case with similar operative findings, with similar result. In 3 cases of gastric ulcer arterial duodenal ileus was noted, but gastro-enterostomy was performed.

There were 5 cases in which the diagnosis was established by x-ray examination only (Figs 462, 463, and 464). One of these had a mobile colon, but the exciting cause of the duodenal obstruction was the traction of several feet of ileum prolapsed into a hernial sac. Radical cure of the hernia abolished the symptoms and x-ray signs of duodenal ileus. The others were treated by an abdominal belt and by the administration of liquid paraffin, which methods have somewhat relieved 3 of them, whilst the others are not improved.

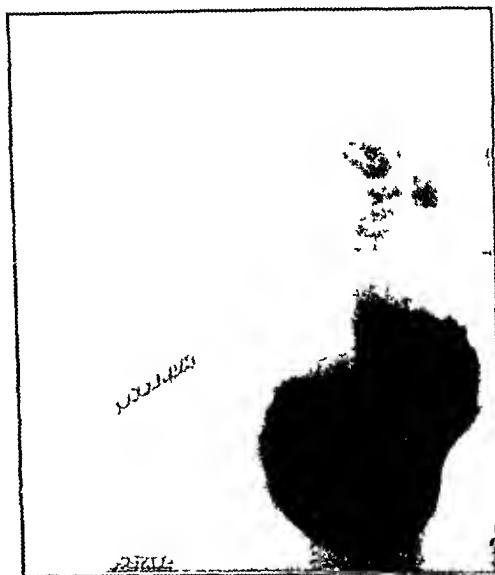


FIG 462—Erect

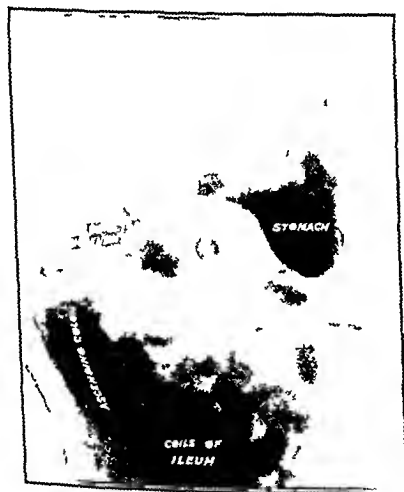


FIG 463—Five hours after meal—Erect



FIG 464—Same case—to show ascent of ascending colon—Six and a half hours after meal—Recumbent

b TRACTION ON THE DUODENUM

The effects of traction of a mobile ascending colon on the duodenum vary according to the type of duodenum that is present. Sometimes the whole of the first stage of the duodenum can be drawn down freely, sometimes it is highly placed and fixed. A strong hepatoduodenal ligament accounts for this fixity in some cases.

We had frequently noted variations in the accessibility of the first stage of the duodenum, but it was not until we read Waugh's paper that we began to realize their significance. An investigation of these variations in infants at birth has been undertaken by Dr C. M. West, University Anatomist, at Trinity College, Dublin, his observations,

which will be published shortly, go to prove that there is a congenital fixed type of duodenum and a congenital mobile type. These results agree with our own in the living subject, and suggest that mobility of the first stage of the duodenum is not secondary to mobility of the colon, nor are the two conditions necessarily associated (*Fig* 465, 466, and 467).

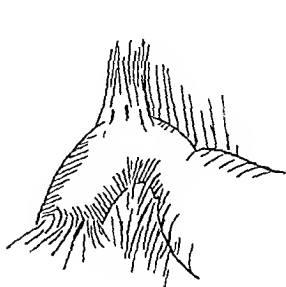


FIG. 465 —Average type

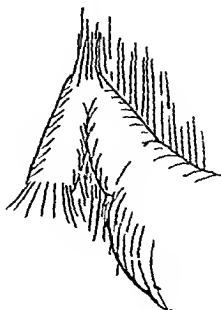


FIG. 466 —Fixed type

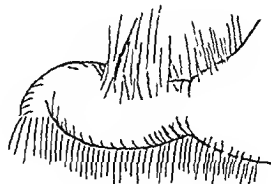
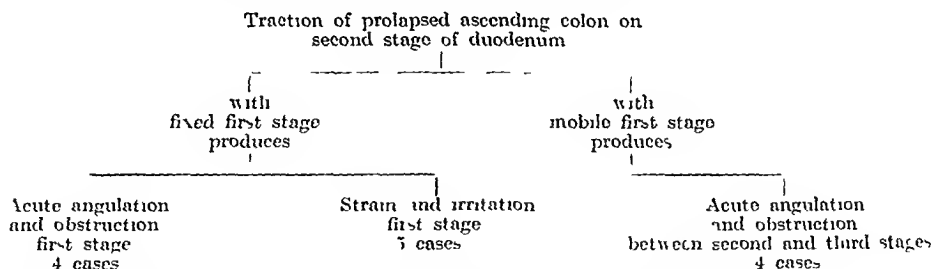


FIG. 467 —Mobile type

Both the mobile and fixed types of duodenum have been found in association with a normally fixed colon.

Second Stage of Duodenum—In the fixed type, traction on the second stage of the duodenum produces either an acute angulation of the first stage, resulting in obstruction, or exerts a strain on the most fixed portion of the first stage, which strain may lead to local inflammatory changes.

In the mobile type, traction on the second stage draws the upper part of this portion downwards and forwards. As the third stage of the duodenum is always fixed, the descent of the first and second stages leads sooner or later to the development of an acute bend at the lower part of the second stage or at its junction with the third stage. The effects of traction on the duodenum may be tabulated thus —



Acute Angulation and Obstruction, First Stage of Duodenum —

Case 1—Female, age 24, of healthy appearance, had an attack of acute epigastric pain and vomiting a year before. The pain came on suddenly and had no relation to food, it radiated to the back and right shoulder and in a few minutes culminated in copious vomiting and prostration. Inside half an hour she felt and looked well again. Exactly similar attacks recurred at intervals of anything from one to fourteen days. If she lay down immediately when the pain began, the vomiting was prevented and the pain relieved. She was always constipated, and took a purgative every morning.

Radioscopy—Dilated stomach large six hour residue. Tenderness over pylorus and duodenum. Caput duodeni well visualized and normal. Ascending colon prolapsed but straight. Excursion of hepatic flexure 3 in., some lateral mobility. Meil reached rectum in twenty four hours.

Operation—Stomach normal. First stage of duodenum highly placed and fixed with sharp angulation accentuated by traction on hepatic flexure of colon. No sign of ulceration. Ascending colon had complete primitive mesentery. Appendix normal. Appendicectomy and colopexy. Result no attack since operation fourteen months ago. Radioscopy a year after. Stomach normal in size shape tone, and position. No six hour residue. Excursion of hepatic flexure 3 in., no lateral mobility.

Acute angulation of the first stage of the duodenum with obstruction was present in three other cases. The pain was, however, related to food coming on from half an hour to an hour and a half after a meal and relieved by recumbency or vomiting. There was no six hour gastric residue, although the stomach was very atonic in two of the cases. The ascending colon was prolapsed in one and angulated in two. These patients have remained well after colopexy for over eighteen months.

Strain on the First Stage of the Duodenum Duodenal Irritation —

The anatomical findings in this group were as follows. The first stage of the duodenum was highly placed and fixed, and traction on the hepatic flexure produced an anemic area at the point of greatest fixation. The angulation was nearly as acute as in the obstructive group just considered, the ascending colon was either of the prolapsed or angulated type, in fact it is impossible to point to any real anatomical difference between the two groups. The radioscopic and clinical findings were, however, quite different and warrant separate classification.

Case 5—Male, age 38. He had suffered for two and a half years from attacks of pain midway between the umbilicus and the tip of the ninth costal cartilage. There was usually an interval of some weeks between the attacks. The pain came on two hours after a light meal and three hours after dinner. It lasted till the next meal, which relieved it. It awakened him about 2 a.m., and was then relieved by a biscuit. The pain was accompanied by a "ipaety rather than an appetite for food." Constipation was always associated with the attack. In the morning he felt heavy and sick. The pain was considerably relieved by lying down on his left side, and could be produced or increased by lying on his right side. From the onset of symptoms he had been under medical treatment, which included everything but the recumbent posture.

A RAY REPORT—Normal shaped tonic stomach. Hypermotility present, the whole meal leaving the stomach in twenty minutes, and reaching the colon in six hours. Dropped mobile hepatic flexure. Angulation of ascending colon. A ray diagnosis of duodenal irritation and mobile hepatic flexure.

OPERATIVE FINDINGS—First stage duodenum highly placed and fixed. There was a patch of congestion at the point of greatest fixation, but no induration was present. It was slightly dilated, as also was the third stage, in which the dilatation stopped at the root of the mesentery. A firm parietocolic fold fixed the ascending colon at the junction of its lower third and upper two-thirds. Appendix normal. Appendicectomy and colopexy. Result after fifteen months "No trouble since."

Case 6—Female, age 29. Patient in 1916 began to suffer from pain in the epigastrium, coming on about an hour after food, and relieved by food. Though food relieved the pain it always gave her a sensation of distention. The pain was of an intense gnawing character, accompanied by nausea and eructations of gas. She was awakened by the pain at 1.30 a.m., and was then relieved by lying on her face with a pillow under the abdomen. She never vomited. When erect she could not bear the pressure of corsets. She had always been constipated. She had been treated for a few months at a time by six doctors, without relief, for five years.

No abnormality of the gastro-intestinal tract was noted on a ray examination. The hepatic flexure had a vertical range of mobility of 2 in. The ascending colon was straight, and could be moved towards mid line.

OPERATION—Stomach normal. First stage of duodenum highly situated and fixed, it was rendered acutely angulated by traction on the mobile hepatic flexure. There was no induration or other sign of ulceration. The third stage was not dilated. The ascending colon had a complete primitive mesentery, and it seemed to be suspended from the fixed first stage of the duodenum. Owing to the high position of the latter, the ascending colon, although mobile, appeared normal on a ray examination. Colopexy. Result, one year after "Feel better than I have done for years."

There were five cases of what we have termed duodenal irritation, of which Cases 5 and 6 are fair examples. The general clinical aspect of the cases was as follows. Pain in the epigastrium from one to three hours after a meal, lasting to the next meal and relieved by it. Three of the patients were awakened by the pain between one and two o'clock in the morning, when two were relieved by eating something, and one by adopting the prone position. Posture had some relation to the pain in every case, but the effect was not so marked as in the other groups. One patient could produce or increase the pain by lying on the right side, another relieved it in the same manner, while the rest found some relief in the prone or supine position. Each attack was the same in the same patient, but the interval between attacks varied considerably, in two there was practically no interval.

The one thing common to each attack in each patient was that it always corresponded to a period of constipation. Four of the 5 cases showed hypertonicity and hyperperistalsis of the stomach, with rapid evacuation of its contents.

More than a year has elapsed since colopexy was performed in these cases, and in none of them have the symptoms returned. In addition to the 5 cases of duodenal irritation just recorded, 5 others with similar x-ray and clinical findings were observed, but were not checked by operation. One of these had been in hospital nine months previously. At that time his symptoms were all referred to the right iliac fossa. At operation an adherent appendix was removed and a strong parietocolic fold divided. This relieved all his symptoms. Soon after leaving hospital he began to develop symptoms suggestive of duodenal ulcer. When he was admitted for the second time the ascending colon was found prolapsed and straight, and the stomach showed hypermotility and rapid evacuation. The former operation had turned an angulated ascending colon into a prolapsed one, and replaced the symptoms of colonic obstruction by those of traction on the duodenum.

The significance of these cases lies in the fact that the treatment that gave them most relief was not alkalinization and not frequent meals, but an abdominal belt and liquid paraffin, two measures which tend, by supporting the ascending colon and reducing its weight, to diminish the strain exerted by it on the duodenum. There were only two cases of definite duodenal ulcer in this series. The duodenum was high and fixed, and the colon mobile. Gastro-enterostomy was performed.

Obstruction between Second and Third Stages of the Duodenum —

Case 7 — Female, age 60 For fifteen months before admission she had suffered from attacks of pain starting in the epigastrium and radiating along the right costal margin. The attacks came on suddenly every four or five weeks, and were ushered in with pain so severe that the patient had to lie down immediately. The pain lasted about fifteen minutes and left the patient prostrate. It had no relation to food. Nausea accompanied the pain, but there was no vomiting. Immediately after the third attack jaundice appeared, this lasted about a fortnight and reappeared after each subsequent attack. The urine contained bile, and the stools were always coloured. The gall bladder was not dilated.

X-RAY REPORT — Normal shaped stomach, good tone. Small barium residue seven hours and a half after meal. No deformity in the region of the pylorus. Ascending colon prolapsed, very mobile in all directions, vertical excursion of hepatic flexure 3 in. Stasis in cecum.

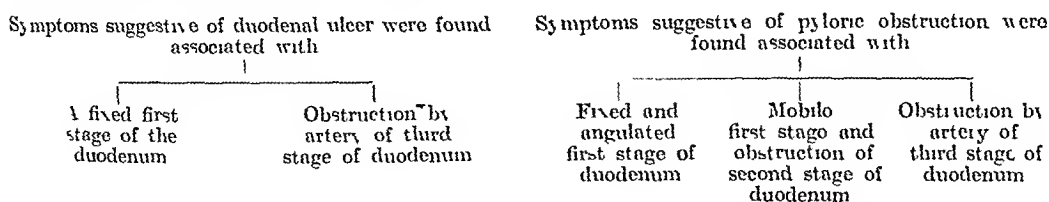
OPERATION — Gall bladder not dilated, no stones palpable in it or in biliary passages. Supra-mesocolic portion of duodenum very mobile and dilated. Slight traction on hepatic flexure brought the first stage of the duodenum below the level of the third. Site of obstruction was in lower part of second stage, and the duodenal deformity apparently produced obstruction of the common bile duct. The ascending colon possessed a complete primitive mesentery. Appendicectomy and colopexy. Result after a year and nine months. "No return of pain or jaundice."

In 3 other cases operation revealed excessive mobility and dilatation of the supra-mesocolic portion of the duodenum without dilatation of the inframesocolic portion. These patients suffered from attacks at irregular intervals, of epigastric pain accompanied by vomiting. The pain was considerably relieved by lying down, and the vomiting in one case was prevented by this posture. There was no jaundice. These patients had the fullest degree of mobility of the ascending colon. Colopexy was performed in each case with complete relief.

The anatomical deformity probably present in these cases, but masked by the attachment of the transverse mesocolon, was revealed in a formalin subject in the Anatomical Department of the Royal College of Surgeons in Ireland, to which Mr A. K. Henry drew our attention. The first stage of the duodenum was very mobile, and could be lifted up readily from the posterior abdominal wall, the upper two thirds of the second stage had a similar mobility. The supramesocolic portion as a whole lay in the horizontal plane, and could be drawn below the level of the third stage of the duodenum by gentle traction on the hepatic flexure. On dissecting away the transverse mesocolon the upper two thirds of the second stage were seen to join the lower third at an acute angle opening downwards. The kink at this point seemed to have been sufficient to produce obstruction, for the proximal segment was dilated whilst the distal was contracted. The common bile duct

entered the duodenum just at the bend, but there was no evidence of biliary obstruction. The ascending colon had a complete mesentery.

Remarks on Duodenal Lesions in General—There were four separate duodenal lesions associated with mobility of the ascending colon: obstruction first stage, obstruction second stage, obstruction third stage by artery, and strain on the fixed first stage, yet there were not four separate clinical pictures, there were two. Speaking broadly, some of the patients presented symptoms suggestive of duodenal ulcer, others those suggestive of pyloric obstruction. The relation of symptoms to the anatomical condition found may be represented schematically thus—



Thus the symptoms revealed not the exact *type* of lesion, but its *site*—the duodenum. The symptoms described under 'duodenal irritation' and in *Cases 3 and 5*, sufficiently resemble those in Moynihan's classical description of duodenal ulceration to be classed as at least 'suggestive of duodenal ulcer'. Moynihan has given us the clinical picture of irritative lesions of the duodenum, and *one* of these irritative lesions is ulceration. All the cases in this series with these suggestive symptoms had something wrong with the duodenum, viz., duodenal ulcer in 1, arterial duodenal ileus in 1, and demonstrable strain on the first stage in 5. The first symptoms in one case of actual ulceration were those of peritonitis from perforation. In short, laparotomy in cases with these symptoms may not demonstrate duodenal ulcer, but it will demonstrate a duodenal lesion, if the *whole* duodenum be inspected.

c TRACTION ON THE GALL-BLADDER

Of the patients with arterial duodenal ileus, 2 had a peritoneal fold continuous with the lesser omentum passing from the gall-bladder to the duodenum and hepatic flexure of the colon. One patient with acute suppurative cholecystitis had the same type of fold. The following case was the only one in the series in which this fold was directly responsible for symptoms—

Case 8—Pensioner, age 46. He had suffered at irregular intervals since 1917 from pain in the right hypochondrium radiating to the right shoulder. The pain came on suddenly, was very severe, and was accompanied by vomiting and cold perspiration. It was relieved by lying down. There was no relation to food. Pressure under the right costal margin opposite the ninth costal cartilage elicited tenderness.

Radioscopy revealed a normal gastro-intestinal tract with no undue mobility of the ascending colon.

OPERATION—A firm band continuous with the lesser omentum, passing from the gall-bladder to the duodenum and continued on to the hepatic flexure, was found. The upper third of the ascending colon had a mesentery, and traction on the colon dragged the gall-bladder downwards. The band prevented the mobile portion of the ascending colon from sagging, and so the latter had appeared normal on x-ray examination. The band was divided. This operation was performed ten months ago, and so far the patient has been without symptoms. Division of the band, however, has probably produced an angulated type of ascending colon.

d TRACTION ON THE PYLORIC PORTION OF THE STOMACH

At operation it is easier to prove that the prolapsed hepatic flexure can drag on the duodenum than that it can drag on the stomach, because the duodenum is relatively fixed whereas the stomach has a certain postural range of movement. Displacement downwards of the proximal third of the transverse colon is limited by the fixation of the normal hepatic flexure. When the flexure is not fixed, downward displacement of

the transverse colon is seen to drag on the stomach and draw its pyloric portion down as far as the gastrohepatic omentum permits. The strain of colonic traction is most marked along the lesser curvature. Variations in the length and strength of the gastrohepatic omentum probably determine whether the maximum strain falls on the stomach or on the duodenum. If the lesser omentum be long the pyloric portion of the stomach can sink, and the weight of the ascending colon falls on the second stage of the duodenum, with the effects already noted. If it be short and strong, it acts as a suspensory ligament not only to the pars pylorica, but also to the ascending colon and part of the transverse. Such a strain may impair the vitality of the gastric wall along the lesser curvature, and be a predisposing cause of gastric ulcer.

Case 9—(Included by the courtesy of Sir Thomas Myles.) Male, age 52. Strong muscular development. Suffered for the last twenty years from attacks of 'roasting' pain in the epigastrium. At first the attacks occurred two or three times in the year and lasted for about three weeks, but as time went on they became increasingly frequent. He was always constipated when the pain made its appearance. For some years the pain was relieved somewhat by a hot drink, but had no other relation to food. *It was always relieved by lying down*, and never came on when he was recumbent. If he stayed in bed he could eat anything without fear of pain. Vomiting could be prevented by lying down. Medical treatment availed nothing unless he stayed in bed, when it was unnecessary. At operation a small indurated ulcer was found on the lesser curvature near the pylorus. The duodenum was normal. The ascending colon had a complete mesentery. Gastro-enterostomy was performed.

It is not probable from the appearance of the ulcer, that it had been there for twenty years, and the characteristics of the pain never changed nor did its relation to posture. The anatomical findings suggest that the mechanical cause of the long-standing symptoms was a factor in the causation of the short-lived ulcer.

There were 6 other cases of actual ulceration of the stomach in all the ulcer was on the lesser curvature, and in 3 it was situated close to the incisura angularis and had produced hour glass contraction of the stomach. The ascending colon possessed a mesentery in each case. As organic stenosis was present in all these cases, fixation of the colon was not attempted. Partial gastrectomy was performed in 2, excision of the ulcer in 1, and gastro-enterostomy in the rest. One case already cited had colopexy performed at a later date for symptoms of arterial duodenal ileus.

c TRACTION ON THE RIGHT KIDNEY

When the hepatic flexure is in its normal relation to the anterior surface of the right kidney, it takes some part in supporting that viscus and in preventing it from slipping down the inclined plane between the last rib and the iliac crest. It is difficult to understand how nephroptosis could occur with a fixed hepatic flexure. In all the cases of movable right kidney in this series the hepatic flexure was freely mobile and was not in apposition to the kidney. When the flexure is mobile it offers no obstacle to the descent of the kidney, and the weight of the ascending colon transmitted through the peritoneal attachments to the renal fascia draws the latter downwards and thus removes a further obstacle. If the mobility of the ascending colon be the cause, or even if it be but an association of movable kidney, most of the gastro-intestinal symptoms ascribed to the latter are readily explicable. Any case might present symptoms attributable to the kidney itself and at the same time those due to variations in the form of the ascending colon or to traction of this segment of the intestine on other structures.

Case 10—Female, age 41 unmarried. For several years patient had suffered from attacks of flatulence and regurgitation of food. The attacks always came on when she was constipated, lasted for a day or more and recurred at irregular intervals. Two months before admission they became more severe. When in bed at night she experienced a dull pain above the umbilicus, accompanied by flulent eructations and a very bad taste in the mouth. She was relieved by sitting up in bed and clasp ing the knees. During this period she began to suffer from a dragging pain in the right loin, accompanied by frequency of micturition. This pain was relieved by lying down, and returned when she got up in the morning, it was increased by exercise. The frequency was due to polyuria. The fingers could be inserted above the upper pole of the right kidney. The stomach was dilated.

Radioscopy demonstrated retention after 6 hours in the second and third stages of the duodenum, and after 36 hours in the ascending colon, which was of the ingulated type

OPERATION—Stomach dilated, duodenum dilated down to crossing of superior mesenteric artery, right kidney freely movable. Ascending colon with complete mesentery, plicocolic fold half way up. Appendix normal. Colopexy was the only treatment adopted. Radioscopy two months after operation revealed no duodenal obstruction, and the right kidney was no longer movable. Two years after, the patient reported 'well'

The long standing flatulence and regurgitation of food, and the epigastric discomfort coming on so constantly during recumbency and so constantly relieved by the squatting posture, can be attributed in the light of the cases described above, to arterial duodenal ileus, the dull dragging pain in the loin relieved by lying down, and the frequency of micturition associated with it, were doubtless due to the movable kidney

Arterial duodenal ileus was the cause of dilatation of the stomach in another case of movable kidney. Both cases were referred to the surgical side because of the local renal symptoms

It is significant that both patients found relief from pain in the manner described fifteen years ago by Newman in cases of movable kidney, namely by sitting up, clasping



FIG 468



FIG 469

the legs, and putting the head down on the knees. Every one of our patients who relieved his pain in this fashion had arterial duodenal ileus

Another patient presented the symptoms of duodenal irritation in addition to those of movable kidney, at operation, traction on the mobile hepatic flexure did not affect the position of the kidney but produced acute angulation of the first stage of the duodenum, which was highly placed and fixed. In these three cases the gastric symptoms were produced by the mobile colon and not by the movable kidney. Each patient has remained free of symptoms for more than a year after colopexy. A movable right kidney may, however, be directly responsible for duodenal obstruction, for there is no doubt that nephropexy alone removes gastric symptoms in some cases. The too frequent failures of nephropexy indicate that the primary cause of renal mobility has not been removed, or that an associated lesion has been overlooked

There were 5 cases of movable kidney with purely renal symptoms, 1 had typical Dietl's crises, 3 had intermittent attacks of dull dragging pain in the loin accompanied by frequency of micturition. In 2 of these hydronephrosis was demonstrated by pyelography (Figs 468 and 469). The first patient has had no crisis since colopexy was performed

seventeen months ago. One case of hydronephrosis was so advanced that nephrectomy was necessary, I was improved temporarily by colopexy, but a year afterwards reported that he was the same as before operation. The fourth case refused operation and the symptoms are unchanged, and the fifth was relieved by an abdominal belt. Of the 8 cases of movable kidney, 3 had well-developed abdominal muscles. None had general enteroptosis.

Line, in 1903, attributed mobility of the right kidney to the drag of the ascending colon, and seven years later this cause of renal mobility was advocated strongly by Longyear in his book on nephrocoloptosis. This author also held that duodenal obstruction could be caused by ptosis of the ascending colon, whether the kidney was mobile or not. As far as we know, he was the first to emphasize the fact that the ascending colon was the primary cause of so many associated lesions.

6 REMARKS ON RADIOSCOPIC TECHNIQUE

The technique adopted in the examination of our cases was as follows. The bowels were cleared by enema on the night previous to the examination, no aperient being given. At 5.30 a.m. an opaque meal was given, consisting of $3\frac{1}{2}$ oz. of barium sulphate, 2 oz. of bread, and 8 oz. of milk. The patient remained in bed until the first examination six hours later. We believe that it is of the greatest importance, in determining the existence of any mechanical obstruction at the pylorus or in the duodenum, that the patient should be recumbent in the interval between the ingestion of the meal and the 6-hour examination, and in cases of this type one of us has made this procedure a routine. A 6-hour retention is abnormal, and in a recumbent patient may almost certainly be regarded as an indication of obstruction at the pylorus or in the duodenum. A hypotonic or atonic stomach may retain a large amount of the meal after six hours if the patient has been allowed to go about and carry on a normal life, but when recumbent the same stomach may empty in the normal time, showing that no fixed obstruction is present. We first screen our patients in the erect posture, and the presence of a residue in stomach or duodenum is noted, also the position of the meal in the ileum and colon. A 6-hour retention in the stomach is regarded as abnormal, and an indication of some degree of obstruction either at the pylorus or in the duodenum. If peristalsis is proceeding, one notes whether the food passes freely through the pylorus, and, if so, whether the delay is occurring in the duodenum. If the 6-hour residue is very small, or no food remains in the stomach, a second meal is given, consisting of barium sulphate suspended in muceilage and a little water added. This fluid mixture we have found to be very suitable for the examination of the duodenum. If constriction of the third stage by the mesenteric artery is present, the accumulation of the opaque mixture proximal to this vessel can readily be seen, and in severe cases of constriction regurgitation or reverse peristalsis can be observed. In some cases we have found that the duodenum is more easily seen when in an erect position, in others the recumbent posture is more suitable. The ability to visualize the duodenum varies greatly with different patients, and one cannot therefore lay down any hard-and-fast rule regarding the best position for examination. In those cases of duodenal ileus which we have observed radioscopically the obstruction was plainly discernible whether the patients were standing or lying down. Plates or films are exposed if required, but in most cases we have found that the maximum amount of information may be gained by radioscopy combined with palpation. In some of our cases a 6-hour gastric residue was found but the duodenal ileus was not recognized although found at operation. We would suggest that failure to detect this condition may have been due to pylorospasm preventing the filling and consequent visualization of the duodenum during the examination.

The examination of the colon calls for no special comment further than to say that it is best seen at the 24-hour observation as a rule, and is already described, the measurements of the cæco ascendens are made with the patient first standing, and then lying down.

7 GENERAL OBSERVATIONS

The relation between symptoms and posture was so definite in these cases that it was impossible to avoid the conclusion that something inside the abdomen changed its position according to the posture of the patient, and by so doing caused or relieved the symptoms. The only structure which reacted abnormally to posture in every case was the ascending colon. Pain was associated with a definite position or deformity of the ascending colon in over 90 per cent of the cases. If the position were altered or the deformity removed, pain disappeared. If an abdominal tumour be palpated in a patient with abdominal symptoms, an attempt is made to demonstrate a connection between the tumour and the symptoms, likewise, when the ascending colon is found deformed or mobile, it is reasonable to regard the clinical picture in relation to that abnormality.

The manner in which the symptoms are produced is primarily mechanical. Symptoms referred to the right iliac fossa were always associated with obstruction in the ascending colon itself. No matter how mobile the ascending colon was, the local symptoms were insignificant or absent unless angulation or collapse was present. Traction of the ascending colon on the duodenum produced some type of duodenal obstruction in several cases, and the symptoms were obstructive symptoms. There was a definite group of cases classed under 'duodenal irritation' in which no obstruction could be demonstrated, but in which the first stage of the duodenum was manifestly subject to strain. In short, with the exception of those present in the cases of duodenal irritation, all the gastro-intestinal symptoms were due to intermittent obstruction in the ascending colon itself or in the duodenum. Acute angulation of the ascending colon—only possible when fixation is faulty—caused obstruction in this region. Acute angulation of the duodenum and tension on the superior mesenteric artery—both produced by traction of the mobile ascending colon—were responsible for the duodenal obstruction. When the abdomen is opened under local anaesthesia, traction on a mesentery seems to be the only thing that causes pain, but such pain in our experience is referred to the back. Hurst⁵ has shown that stretching or distention of the intestine causes pain, and when operating on cases of acute obstruction under local anaesthesia we have noted that pain coincides with peristalsis. Stone in the common bile-duct and ureter cause pain apart from mesenteric traction. In other words, although traction on a mesentery does cause pain, it is not the only cause, and these cases suggest that it is not the chief element in the cases of mobility of the colon.

Although an ascending mesocolon is a congenital defect, yet many years may elapse before abdominal symptoms appear, and after their onset there may be lengthy periods of immunity. Some exciting factor precipitates the owner of a mobile colon into invalidism. In the cases of *Class 2* the exciting cause was often accumulation of faecal masses in the ascending colon, the weight of which was thereby increased. In *Class 1* the proximal colon sooner or later failed to compensate for the obstruction introduced in its course. A cervical rib is a congenital abnormality, yet the symptoms do not appear till some other factor—probably weakness of the musculature of the shoulder-girdle—makes its appearance.

The cases cited under 'duodenal irritation' indicate that a strain on the first stage of the duodenum is caused by the mobile colon.

Reeves, of the Mayo Clinic, has demonstrated that the arteries supplying the lesser curvature of the stomach and the first stage of the duodenum have certain peculiarities which render the blood-supply of these regions relatively deficient, and W. J. Mayo has shown that in anæmic area can be produced on the first stage of the duodenum by traction on the stomach.

In three cases with the fixed type of duodenum a similar anæmic area was produced by traction on the hepatic flexure of the colon. It is possible that the drag of the mobile colon may impair the blood supply of the first stage of the duodenum when the patient is erect and thus be the predisposing factor in duodenal ulceration. The relation of infection and hyperchlorhydria to such a mechanical factor would be a subject for fruitful investigation.

No physician prescribes ambulatory alkalinization in cases of duodenal ulcer, he puts the patient to bed. The recumbent position frequently takes the strain off the duodenum, and is probably as much responsible for a symptomatic cure as the raw eggs, milk, or sodium bicarbonate.

Fifty per cent of the cases here reported were males. The youngest patient was six and the oldest sixty-five.

The aim of this study was twofold: first to decide from our own experience whether the mobile ascending colon could be responsible for abdominal symptoms or not, and next if responsible, to discover the exact anatomical manner in which it produced these symptoms. We have therefore confined ourselves to the statement of clinical states and operative findings. The good result of any operation depends on two factors: (1) That the operation is indicated, (2) That it is properly performed. Unless both factors are known, results cannot be appraised. We have not performed colopexy unless a definite anatomical connection could be traced between the mobile ascending colon and the symptoms. This connection furnished the indication for the operation, and this indication remains, whatever the results. It is with the indication for the operation that this communication deals. Some of the operations were not properly performed. In one case—the first—a carbolic swab was placed in the wound after faulty closure of the peritoneum, and caused irritation of the peritoneal surfaces, this patient was re-operated on twice for adhesions to the abdominal scar. In another case we failed to get adequate relaxation of the abdominal wall, and fixation of the colon could not be performed satisfactorily. Radioscopic examination six months afterwards showed that the colon was as mobile as ever, but whereas it had been angulated, now it was straight. The indications for colopexy can be learned by observation, the technique can be acquired by experience.

8 GENERAL TREATMENT

These cases are orthopædic cases in the modern acceptance of that term, and the treatment necessary is orthopædic. The principles of treatment are —

- 1 To make the mobile ascending colon straight

- 2 To prevent it exercising traction on the structures to which it is attached

We have failed to carry out the first principle by any non-operative method. An abdominal belt properly applied can keep the whole ascending colon up, but it cannot make it straight, angulation and collapse are accentuated.

Administration of suitable purgatives may facilitate the passage of the cæcal contents in spite of the colonic deformity, and intestinal antiseptics may partially inhibit cæcal putrefaction, but these methods are strictly palliative and applicable to the effects, not to the cause. By operation the ascending colon is first rendered straight and is then fixed in a normal position. When the symptoms are due to traction of the ascending colon on other structures, much relief may be given by non-surgical measures. The load of the ascending colon is lessened by suitable diet and laxatives, and thus its weight is diminished. An abdominal belt supports the ascending colon and prevents traction on its peritoneal connections. These measures may relieve the symptoms as a truss relieves a hernia, but they do not cure the patient. Colopexy alone does that.

Of the 76 cases in this series 58 were operated on, colopexy being performed in 44. There were 14 cases in which the ascending colon was observed at operation but in which it was not considered advisable to fix it. These are reported in the body of the paper. Eighteen cases were not submitted to operation, but were treated by the palliative measures outlined above. All but 6 of the total number had undergone medical treatment for periods varying in length from three months to ten years and all were referred to us by physicians.

The results may be summarized generally thus. Non-operative treatment on orthopædic lines gave better results than medical treatment and surgical fixation of the colon better than either.

SUMMARY OF CASES

ANATOMICAL FINDINGS	NO OF CASES	COLO PRAX	RESULTS OF COLOPRAX	OTHER OPERATIONS	NO OPERATIONS	NON OPERATIVE RESULTS	
						Improved	Not Improved
Deformity of ascending colon	18	11	11 well after a year	Appendicectomy and Lane's kink 1	6	2	4
Arterial duodenal ileus	24	14	1 died, 10 well after a year, 3 well after six months, 1 slight pain and stiffness in right loin	Laparotomy 2 Gastro enterostomy 3	5	3	2
Obstruction at 1st stage of duodenum	4	4	3 well after a year, 1 not improved	—	—	—	—
Strain on 1st stage of duodenum	10	5	4 well after a year 1 slight return of old pain in spring and autumn	—	5	4	1
Obstruction at 2nd stage of duodenum	4	4	Well after a year	—	—	—	—
Traction on gall bladder by band	1	1	Well after ten months	—	—	—	—
Movable kidney	8	5	4 well after a year 1 case of hydro nephrosis not improved	Nephrectomy 1	2	1	1
Gastric ulcer	7	—	—	Direct treatment of ulcer and gastro enterostomy 7	—	—	—
Total	76	44	1 died, 2 not improved, 1 improved but not cured	14	18	10	8

We have been exceptionally fortunate in the opportunities given us by our medical and surgical colleagues of the Richmond Hospital, who have allowed us to observe their cases and have given us full liberty in investigation. We wish to thank also all the students and nurses who submitted to radioscopic examination, without their help we could not have undertaken the inquiry. We are indebted to Dr E C Smith for several of the diagrams. Professors A F Dixon and E J R Exatt have most kindly given us every facility in their respective Anatomical Departments. The influence of Waugh's inspiring paper⁶ on our work is obvious.

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A CONTRIBUTION TO THE SURGICAL TREATMENT OF ATONIC DYSPEPSIA

By CHARLES A. PANNETT, LONDON

IN the continued study of cases suffering from gastric disorders, it becomes remarkable how much subjective sensations depend upon motor upset rather than upon secretory derangement. A man may go through life unaware of the fact that his stomach secretes little or no hydrochloric acid and no enzymes, but he can hardly live a completely comfortable existence if the emptying time of his stomach is delayed very much beyond the normal period of evacuation. Equally striking, when a review of a number of clinical records of gastric cases is made, is the fact that neither by the history of the illness, the results of chemical examinations, nor the changes in the appearance of the stomach cavity as visualized by Röntgen rays, can a correct diagnosis be arrived at infallibly.

A group of cases exists where, with an approximately normal secretion of juice, there is a long delay in the emptying of the stomach as shown by the barium meal. In the recrudescence and subsidence of symptoms, and in other aspects of the clinical picture, there is a very close mimicry of gastric ulcer with pyloric stenosis, yet at operation, to naked-eye inspection, no lesion can be detected in the stomach, duodenum, gall bladder, or appendix. There is an alteration in the motor functioning of the stomach which may well be dependent upon some disturbance of the nervous mechanism by which it is controlled. On this supposition E. Bireher¹ conceived the idea of cutting off both the inhibitory impulses which travel to the general body of the stomach, and the contraction exciting impulses which go to the pyloric ring, by section of the vagus nerves. Thus he did by dividing all the branches he could see near the lesser curvature, both on the anterior and posterior walls. He published a series of cases which showed remarkably favourable consequences of the operation. M. A. Latarjet² made a study of the nerves of the stomach and found that the branches of the vagus contain sympathetic fibres which reach them by anastomotic paths from the celiac plexus. He devised a slightly different procedure for section of the nerves. His investigations really show that Bireher's method in effect cuts off both vagal and sympathetic impulses. As long ago as 1886, F. Hofmeister and E. Sehntz³ demonstrated that co-ordinate peristaltic movements can take place in an excised stomach in saline solution. Two other observations in this connection are worth remembering. Stewart and Barber⁴ showed that in normal dogs after sleeve resection the distal segment of the stomach exhibited more powerful and regular peristalsis, whilst W. J. Mayo⁵ has observed a similar phenomenon in man after a gastric ulcer has been removed by this method. In both these instances the distal part of the stomach is almost completely cut off from nervous impulses of central origin. W. B. Cannon⁶ was able to cut off nerve impulses going from the duodenum to the pylorus by making a circular incision of the duodenal wall down to the mucosa without penetrating the lumen. It seems probable that a similar operation on the stomach, at the junction of the fundus with the body, might also deprive the main part of the stomach from centrally arising nerve impulses, mainly inhibitory, and do this more effectively than by either Bireher's or Latarjet's method. This belief was tested in the patient whose clinical record is here reported. However, Barber⁷ had already tested the effect of this operation in the normal stomach in animals and found that more powerful pro- and anastaltic waves resulted in the pyloric region in fact the effect was indistinguishable from that which he obtained by thoracic section of the vagi.

Case—A H, male, age 46, had suffered from intermittent attacks of pain in the right side of the epigastrium for eighteen months. The time relation to the taking of food was variable, half an hour to two hours. The pain was relieved by taking warm milk. Vomiting was a prominent feature of the attacks. Careful treatment in the medical wards had failed to bring him any relief. Other abdominal signs than some spasm of the upper recti and tenderness in the epigastrie angle were wanting. The fractional test-meal was more interesting. It showed (1) A large volume of resting contents with high acidity and a moderate amount of organic acids, (2) Marked hyperchlorhydria (hypersecretion) throughout digestion, (3) Starch present in the stomach at the end of 2½ hours. The curve is shown in the chart (*Fig 470*)

The radiographic examination showed that there was a dilated dropped stomach, with a large residue still left after eight hours (*Figs 471-475*), so that organic obstruction at the pylorus seemed a safe diagnosis.

At the operation no evidence of ulcer in the stomach or duodenum was discovered. The gall-bladder had a healthy appearance, and the appendix was a normally small pale organ with no sign of inflammation or constriction. A circumcision of the stomach was made as near to the cardiac end as could conveniently be done. The incision was carried

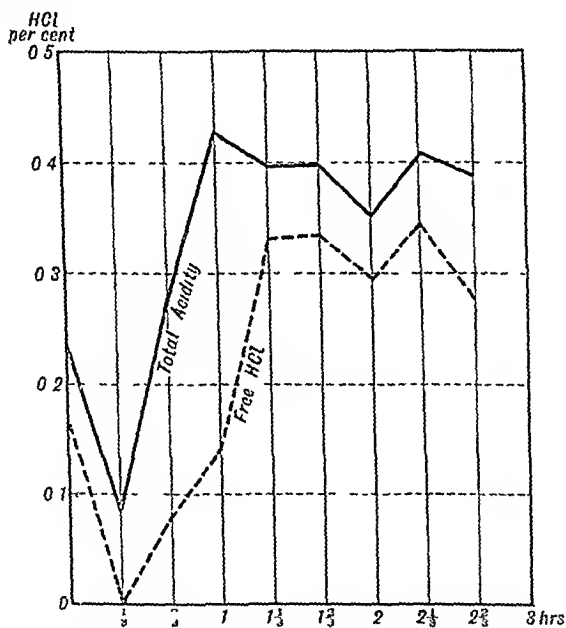


FIG 470—Chart showing result of the test meal

down to the mucosa all round, except for a very small area on the greater curvature where the left gastro epiploic artery was preserved intact, it went right across the lesser curvature, where the coronary artery was divided between ligatures. The posterior wall of the stomach was reached by traversing the great omentum, and the incision of the musculature was sewn up by a continuous catgut suture. Convalescence was quite uneventful and smooth. Three weeks after the operation, another barium meal was given, and care was taken to follow the identical technique used in the first examination. The plates show quite clearly a diminution in the size of the stomach and a much more rapid emptying, no residue in six hours in place of a large one in eight hours (*Figs 476-479*). Even in four hours the residue is not very large. In addition, the patient affirms that all his subjective symptoms of distress after food have disappeared.

Sufficient time has not elapsed to investigate the ultimate results of this operation. A number of factors may work to vitiate the primary beneficial effects. We do not know whether a stomach deprived of all central control can permanently function efficiently, though we suspect that it can from the results of sleeve resection. Again it is possible that the nerves may regenerate and the vicious functioning be re-established. There will

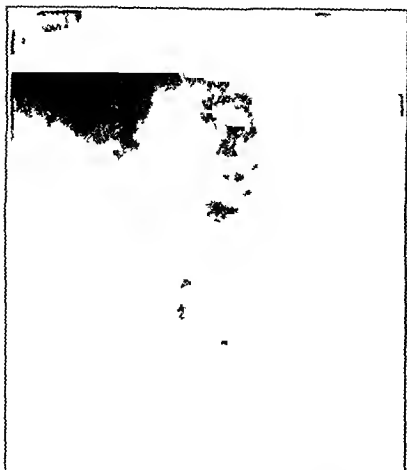


FIG 471 —Before operation 10 minutes plate



FIG 472 —Before operation 2 hours plate



FIG 473 —Before operation 4 hours plate



FIG 474 —Before operation 6 hours plate. Note that a large part of the middle lobe is not on the plate



FIG 475 —Before operation 9 hours plate

certainly have to be a careful selection of cases if the operation is to benefit them for it is done on the supposition that inhibitory impulses to the will of the stomach pass out from the central nervous system in a greater stream than normal, whilst relaxation of the pylorus is prevented by the same nervous efflux. The outstanding features in this patient were an unusually profuse acid secretion, combined with an inability of the stomach to discharge its contents



FIG 176—After operation 1 minutes plate



FIG 177—After operation 2 hours plate



FIG 178—After operation 4 hours plate



FIG 179—After operation 6 hours plate

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A CASE OF DIAPHYSIAL ACLASIS

By A. H. SOUTHAM AND R. S. PATERSON, MANCHESTER

THE term diaphysial aclasis must be considered to be a sufficiently broad one to cover a number of disorders of growth of the diaphysis and this atypical case of bone deformity is therefore included under this title.

The various disorders of growth affecting the skeletal system are of considerable interest owing to the diversity of forms under which they may be met with. The condition may be part of a general constitutional disease such as is seen in rickets, where practically every bone in the body may be affected, or it may be located to certain parts of the skeletal system as in achondroplasia, where only those bones formed in cartilage are affected. It is now considered that many of these diseases are due to some disturbance of function of the glands of internal secretion.

The following case appeared worthy of record on account of the unusual features of the condition, and the limitation of the bony changes almost entirely to the distal segments of the limbs.

History—Thomas E., age 14, was brought to the out-patients' department of the Manchester Royal Infirmary by his mother for 'deformed legs'. The family history showed that the mother had borne thirteen children, eleven of whom were dead. The only other surviving son was in the army and said to show no bony deformity.

Clinical Features—The boy walked well and appeared fully intelligent. He was markedly stunted in height for his age, as is well shown in the photograph (*Fig. 480*) measuring only 3 ft 7½ in., the average height at his age being 4 ft 11 in. The forearms and legs were considerably deformed and shortened. The humerus measured 8.5 in., while the radius was only 3.5 in. in length. The enlargement of the diaphysis was very marked at the wrist. The fingers appeared stunted and thickened, the index, middle, and ring fingers all being equal in length. The femur measured 13 in., but the fibula was only 6 in. long, and the diaphyses at the ankle were markedly enlarged. The head and trunk showed no deformity or abnormality but the sexual characteristics were distinctly in abeyance for his age. The Wassermann reaction was negative.

X-ray Appearances—Skiagrams of almost every bone were taken and on examination of these it was found that the changes present were confined to the forearm, leg, and some of the bones of the hand and that the changes were bilateral. With the exception of the crests of the ilia, all the other bones were found to be comparatively normal.

The shafts of the radius and ulna were of fairly normal width but of greatly reduced length as in achondroplasia (*Fig. 481*). Marked changes were present in the ends of the bones. The epiphyses were small and somewhat irregular but the most pronounced changes were to be observed in the diaphyses. In all the bones affected, the diaphyses



FIG. 480

were large and cup-shaped with numerous bony excrescences and irregularities. The diaphysial line of dense bone so often seen in achondroplasia, was not present, nor was the space between the epiphysis and diaphysis increased, as in rickets, although the relatively large and cupped diaphyses were suggestive of that disease.

Some of the bones of the hand showed the curious deformity of a double epiphysis, there being an epiphysis at each end of the first metacarpal bone, and one at each end of the proximal phalanx of all the fingers.

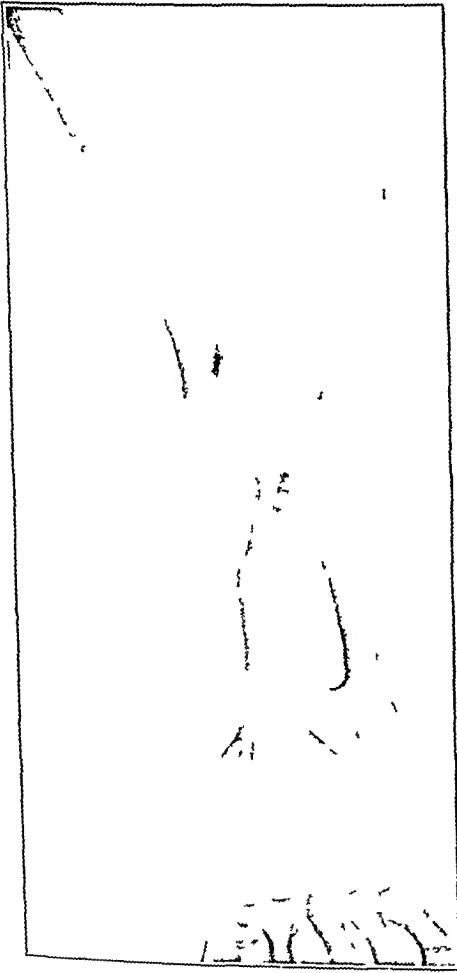


FIG 481

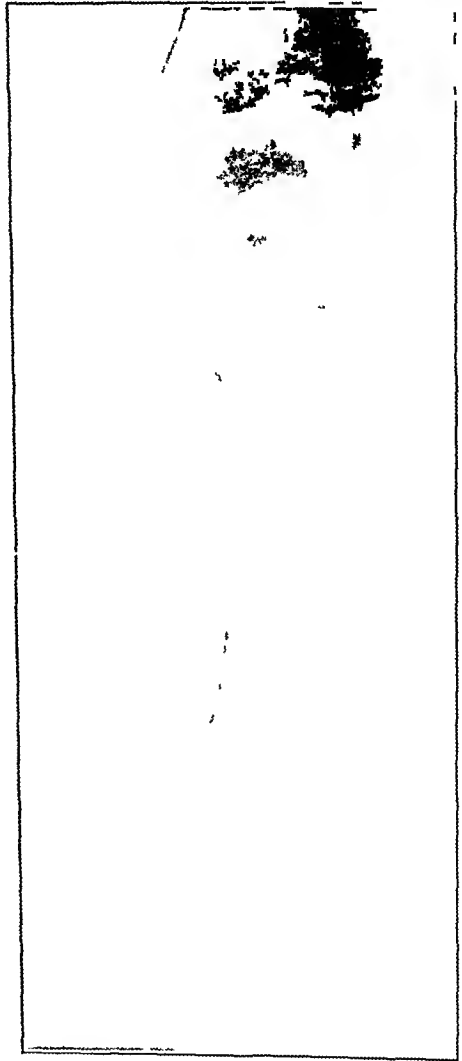


FIG 482

Examination of the bones of the leg (*Fig 482*) showed changes very similar to those described above as occurring in the arm, the diaphysial exostoses at the upper end of the fibula were particularly well marked. There was relative shortening of the fibula compared with the tibia, and of the ulna compared with the radius, in the words of Keith, "the radius becomes a bent bow, the ulna serves as its taut string." Similar bony proliferations were also present at the crests of the ilia.

The skiagrams of this case have been examined by Professor Sir Arthur Keith, and he has kindly reported on them as follows —

The *x* rays show beyond a doubt that this is a true case of diaphyseal aelasis (multiple exostoses).¹ Diaphyseal aelasis is really a disease or growth disorder of cartilage. So are achondroplasia and multiple enchondromata. There is a relationship between these conditions; they run into each other. In this case the skull at its base shows none of the signs of achondroplasia. There are cases—as in the dachshund—where achondroplasia is confined to the limbs, and this appears to be a case of diaphyseal aelasis with a leaning to achondroplasia. Diaphyseal aelasis varies in its manifestations according to the date of onset.

This condition is a disorder of growth where the main disturbance falls upon the modelling of the shafts of the bones. Bones formed entirely in cartilage are free from any disorder of growth—e.g., the tarsal and carpal bones, the vertebrae, and the sternum, likewise bone formed in membrane, the bones of the cranial vault and face. The condition affects the growing ends of the bones, and is also seen along the crest of the ilium and the vertebral border of the scapula. These features are well marked in the case here described.

In achondroplasia the growth of the bones of the arm and legs is defective, the limbs appear stunted, and the stature is diminished. The bones of the trunk are normally developed as in diaphyseal aelasis, as is also the vault of the skull. The base of the skull, being of cartilaginous origin, undergoes premature synostosis, this feature is absent in the case we record.

The interesting features in this case are the limitation of the abnormalities almost entirely to the distal segments of the limbs, and the reduplication of the epiphyses of the phalanges. The examination shows that it has many points in common with the condition described and designated by Sir Arthur Kerth as diaphyseal aelasis.

The skiagrams accompanying this article are of course composite photographs, done to show the relative appearances of the limb bones. Care was taken in obtaining them to eliminate distortion as much as possible and the relative lengths of the bones are approximately correct as compared with the measurements taken on the subject.

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THE CLINICAL ASPECTS OF BRANCHIAL CYSTS

By HAMILTON BAILEY London

INTRODUCTION

THE study of branchial cyst is at once of morphological interest and surgical importance. In this paper the latter side of the question alone will be considered.

In May, 1855 (twenty years after Rathke's startling discovery of the existence of the branchial clefts in mammalian embryos), Lingenbeck¹ described two cases of cyst of the neck in young adults. One was incised and the other treated by the insertion of a seton. In both, gruel-like material continued to discharge, which on examination was found to be rich in cholesterol. He considered these cases to be examples of persistent branchial remnants. Three years later Virchow² described a cyst of the neck which was excised and found to contain epidermal scales. Shortly afterwards three kindred cases were added from Volkmann's³ clinic. From time to time other observers—notably Senn⁴—recorded cases and gradually branchial cyst became an established clinical entity.

It is important to recognize this condition. Branchial cyst is not infrequently confounded with tuberculous cervical adenitis, as the following case summaries show.—

Case 1—Farm labourer, age 17. For two years had a painless lump in his neck. He was told he had tuberculous glands. The swelling was aspirated seven times. Eventually a branchial cyst the size of a Tangerine orange was shelled out.

Case 2—City girl, age 20. For two years had a swelling in left side of neck, which was aspirated five times. She was given a course of tuberculin and advised to keep in the open air. Later, a branchial cyst the size of a hen's egg was dissected out.

Case 3—Milliner, age 27. For the past twelve years has had a lump in the neck. Ten years ago she attended a homœopathic institution and has been an occasional out-patient ever since, during which time the swelling has been aspirated no less than fifteen times. Branchial cyst, size of a Seville orange, easily dissected out.

Case 4—Stoker, age 35. Two years ago, while at sea, a lump appeared in neck. The 'abscess' was incised by the ship's doctor. On return to home port most of his teeth were extracted. The swelling returned and has persisted. Lately it has increased in size. Large branchial cyst dissected out.

Case 5—Female shop assistant, age 20. Eighteen months history of a swelling in neck. Aspirated five times. 'Pus' returned sterile. Given inoculations of tuberculin and ordered oil and malt. Later, a branchial cyst the size of a hen's egg was dissected out.

Case 6—Coal heaver, age 26. Fourteen months has had a painless swelling in neck, getting larger. He was told it was tuberculous. The swelling was aspirated four times and injected with medicated fluid. Lost his employment eight months ago because employer thought the 'neck might burst'. Branchial cyst, size of Seville orange, dissected out. (See Fig 489.)

All the above cases were confirmed histologically.

It is easy to understand how this error arises. Tuberculous abscess is a far more common condition. Moreover, if an aspirating needle be thrust into a branchial cyst, the fluid withdrawn simulates tuberculous pus very closely. The specimen is naturally sent for bacteriological confirmation. But whether it be tuberculous pus or branchial fluid, the "cultures are sterile and no tubercle bacilli seen." Thus the clinician, strengthened by the bacteriological report, may begin seriously to treat a case of branchial cyst as tuberculous abscess.

The diagnosis of branchial cyst is not an academic triumph only. The possessors of these stigmatic remnants are usually in the prime of life, and the stigma of tuberculosis is a heavy burden. Furthermore, if a branchial cyst be incised, there is every possibility of converting the cyst into a fistula, which continues to discharge and is the seat of recurrent attacks of inflammation, rendering subsequent removal difficult.

It will be my endeavour to show that diagnosis can usually be made with precision, and confirmed scientifically.

STRUCTURE

The Wall and its Contents—Gask and Wilson state that branchial cysts are lined by columnar epithelium and contain a glairy mucous fluid. That such a condition exists there can be no doubt.

The records of necropsies in the Pathological Institute of the London Hospital contain many instances of cysts in relationship with the pharynx, lined by columnar epithelium, which have given rise to no symptoms during life.

But branchial cysts lined by columnar epithelium and filled with glairy mucus seldom give rise to symptoms. I have examined the notes of 63 unpublished examples of cysts diagnosed as branchial and removed at operation. In 42 the wall of the cyst was examined histologically and in only one was columnar epithelium found. To discover

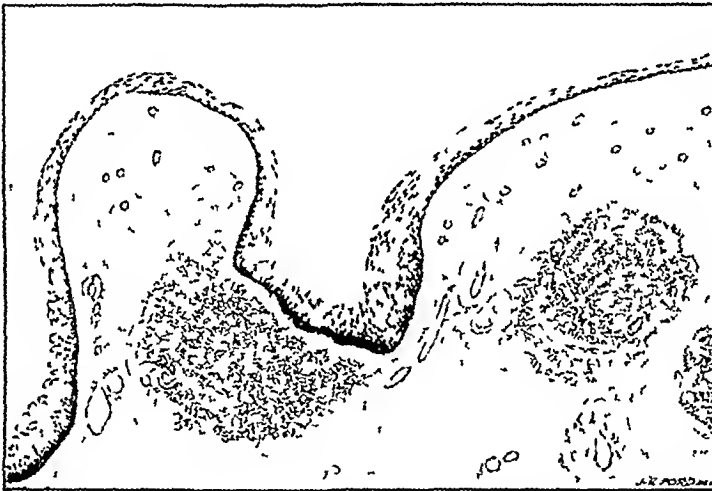


FIG. 483.—Typical section of the wall of a branchial cyst removed surgically showing stratified squamous epithelium on a basis of lymphoid tissue.

In a concrete case of an extirpated mucous branchial cyst it is necessary to search the literature—often to be rewarded by finding a cystic hygroma. Probably, therefore, the existence of the mucous branchial cyst has been established by observations in the post-mortem room rather than by investigations upon the living.

The typical lining of the cyst which gives rise to symptoms is squamous epithelium. The wall of the cyst whether lined by squamous or columnar epithelium, is surrounded by lymphadenoid tissue (*Fig. 483*). Macroscopically the contents of the cyst lined by squamous epithelium is an opaque fluid of such a consistency that it readily passes through an aspirating needle and often through a hypodermic needle. The similarity to tuberculous pus is striking. If, however, such branchial fluid be placed in a dish and moved to and fro the shimmer of its lipid contents will be noticed. The contents of the cyst will again be referred to (on page 571).

RELATIONSHIP TO SURROUNDING STRUCTURES

The diagrams (Figs 484-487) show the relationship of branchial cysts to the surrounding structures, and they are of four types —

Type I—Does not extend deeply. It lies at the anterior border of the sternomastoid, beneath the cervical fascia.

Type II—Passes down to and lies on the great vessels. The wall has to be carefully separated from the internal jugular vein—to which it is varying adherent. This variety appears to be the commonest.

Type III—Extends inwards to the lateral wall of the pharynx. From a theoretical consideration Frazer⁵ states that a large branchial cyst should pass behind the carotid and in front of the vagus. However, it appears from the practical standpoint that the cyst passes between the external and the internal carotid—an observation also noted by Cunningham⁶ Morrison Watson,⁷ Jefferson⁸ and others. In addition, a prolongation of the cyst passes upwards as far as the lateral mass of the atlas, and even to the base of the skull.

The spinal accessory nerve is a very constant posterior relation in *Types I* and *II*.

It may here be stated that at operation the overlying portion of the sternomastoid has been repeatedly observed thinner and flattened out over the cyst (*Types II* and *III*). This point will again be referred to.

Type IV—Is the columnar-lined cyst and is considered

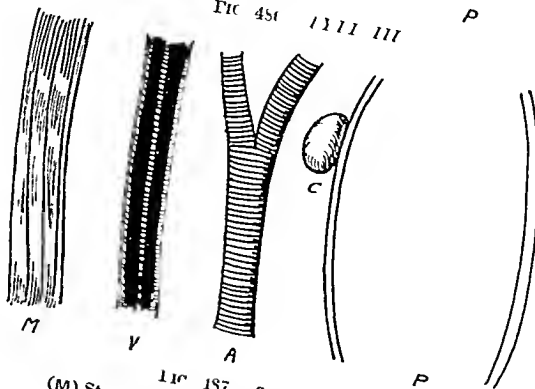
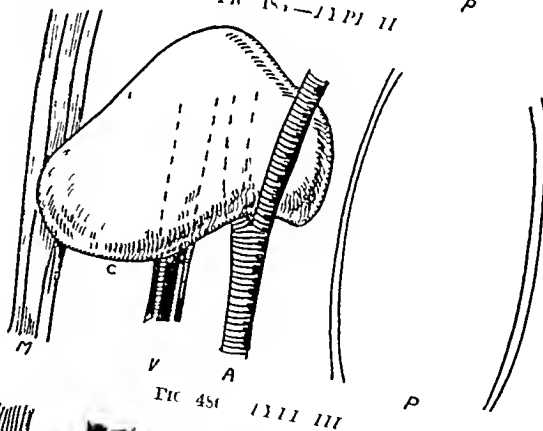
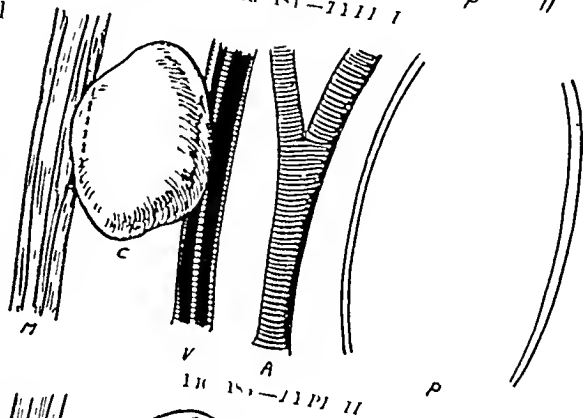
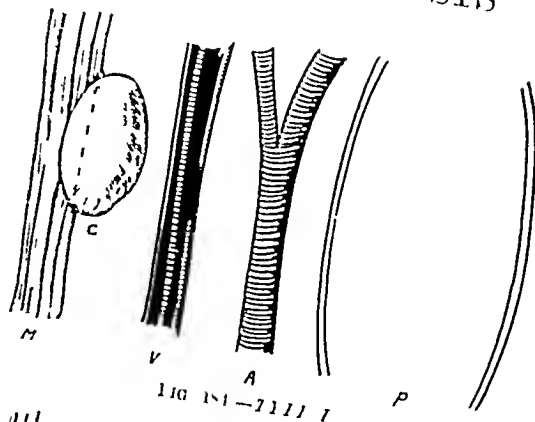


FIG 484 — TYPE I
FIG 485 — TYPE II
FIG 486 — TYPE III
FIG 487 — TYPE IV
(M) Sternomastoid (C) Cyst (V) Jugular vein
(A) Carotids (P) Pharynx

THE BRITISH JOURNAL OF SURGERY

THE PHYSICAL SIGNS ELICITED IN EIGHT CASES (1922-1923)



FIG 188—Age 20 Ten years history painless swelling increasing in size Swelling size of Jaffa orange appearing around anterior border of sternomastoid cystic non translucent No cervical adenitis



FIG 189—Coal heaver age 26 Fourteen months history painless swelling size of Jaffa orange appearing around anterior border of sternomastoid cystic non translucent No cervical adenitis

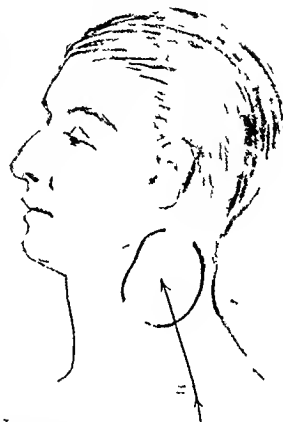


FIG 190—Age 15 Four months history swelling appeared after bicycle accident Swelling size of Seville orange appearing around posterior border of sternomastoid cystic non translucent No cervical adenitis

FIG 191—Age 34 Had a small lump in neck as long as she can remember for six years it has been increasing in size Swelling size of Seville orange appearing around anterior border of sternomastoid cystic non translucent No cervical adenitis



FIG 192—Coal heaver age 10 Twelve months history painless swelling Swelling size of hens egg appearing around anterior border of sternomastoid cystic non translucent No cervical adenitis



FIG 193—Clark age 13 Three years history painless swelling size of fair orange appearing around anterior border of sternomastoid cystic non translucent No cervical adenitis



FIG 194—Labourer age 21 Twelve months history painless swelling size of Seville orange appearing around anterior border of sternomastoid cystic non translucent No cervical adenitis



FIG 195—Age 16 Two and a quarter years history painless lump in neck Lump size of hazel nut just behind angle of jaw movable Diagnosis: parathyroid tumour

COMPLICATIONS

1 *Inflammation*—Occasionally the non-aspirated cyst is the seat of recurrent attacks of subacute inflammation. Exceptionally suppuration occurs.

Three cases of chronic inflammation in the cyst wall were noted in the pathological reports.

2 *Fistula Formation*—Accidental or intentional incision of the cyst is likely to result in (acquired) branchial fistula.

3 *Branchiogenetic Carcinoma*—The existence of branchiogenetic carcinoma is in the opinion of many, not proven. In some clinics it is a recognized diagnosis. On the other hand no less an authority than Sir John Bland-Sutton⁹ states that cancer arising in remnants of the branchial clefts is pure fiction. The whole question cannot be dealt with here. All will agree that branchiogenetic carcinoma as a diagnosis is a last refuge. It can only be entertained after a fruitless search of the mouth, nasopharynx, extralaryngeal recesses, and external auditory canal for a primary growth.

DIAGNOSIS

Speaking generally, the making of a surgical diagnosis resolves itself into seven stages—usually not more than three or four of these will be found necessary.

1 The taking of a history and the general observation of the patient.

2 The elicitation of physical signs.

3 A mental process on the part of the surgeon whereby 1 and 2 are sifted and correlated, and a logical conclusion is drawn.

4 A differential diagnosis is entertained—also a mental process—largely one of exclusion, but reinforced when possible by further physical signs.

5 A scientific confirmatory test—usually performed by a colleague—e.g., a clinical, chemical, bacteriological, histological examinations.

6 The more accessible parts of the interior are rendered visible by ingeniously constructed tubes such as the cystoscope, sigmoidoscope, œsophagoscope.

7 An exploratory operation is performed.

If a diagnosis is still found wanting after the seven stages and combinations thereof have been exploited, there remains but one last court of appeal—the post-mortem room.

The seven stages, which may be termed ‘the surgical crescendo’ will now be reasonably applied to the case of branchial cyst.

1 The Taking of the History the Clinical Features—

The complaint of the patient is the swelling, very rarely is pain a feature of the condition. In only one case was dysphagia mentioned. Not infrequently the patient states that the swelling varies in size from day to day. A few stated that the cyst became tense on occasions—and it was in these that some pain was complained of. Occasionally there is a history of injury.

Sex—Males and females are probably equally affected, although in this series it was very slightly more common in women.

Age—The average age at the time of seeking relief was 23. Sixty-five per cent of cysts were between 17 and 30. Eight were over 40 when they came for advice.

Length of the History—Is most frequently between 1 year and eighteen months. Light had noticed the lump for more than 10 years.

2 The Elicitation of Physical Signs—

Palpate the swelling

Size—This varies, but rarely attracts attention until it has reached the size of a hen's egg.

The Swelling is Cystic—Occasionally, when the cyst is tense (as in the case of the breast and thyroid), it is difficult to elicit fluctuation. In these cases the lump is ‘fixed’ by an onlooker, and fluctuation again sought for in two planes at right angles to each other.

*Position**—The cyst occupies a very constant anatomical position so far as I have been able to ascertain from case histories and personal observation. It lies in relation with the deep surface of the upper half of the sternomastoid, or some part thereof. It nearly always protrudes around the anterior border of this muscle, very occasionally around the posterior border, as *Fig. 490* shows. Most commonly its centre is opposite the great cornu of the hyoid bone. These observations almost without exception coincide with physical signs of reported cases in the literature.

It is of great importance to determine the relationship of the swelling to the sternomastoid. It has already been pointed out that this muscle is thin and flattened out over the cyst. Consequently by mere palpation it is sometimes impossible to make out its relationships, unless the muscle be rendered taut.

Stand behind the patient. Ask him to push his chin as hard as possible against the palm of your hand. This makes the sternomastoid very tense. With the other hand palpate the sternomastoid from below (where it is normal) upwards, paying special attention to the anterior border.

Translucency—In this series there is no record of a translucent branchial cyst. But it is possible that those rare cases springing from the pharyngeal end of the cleft, lined by columnar epithelium and filled with mucus, having attained considerable dimensions, are translucent.

3 *The Facts Collected and a Deduction made therefrom*—Here is a patient, age 23 who for eighteen months has had a cystic painless non-translucent swelling situated beneath the upper half of the sternomastoid but appearing around its anterior border. It is possible that this is a branchial cyst. A diagnosis can never be made at this stage. It is only a guess.

4 *Differential Diagnosis*—

i *From Breaking down Tuberculous Glands*—Palpate the neck for enlarged glands. In order that no glands be overlooked, it is well to have a routine which scrutinizes every cervical lymphatic group. A useful order with a march of sequence is—

Standing behind the patient (whose head is bent slightly forward to relax the musculature), palpate (a) submental (b) submaxillary (c) jugular chain, (d) supraclavicular, (e) posterior triangle, (f) posterior auricular (g) pre-auricular.

After this the possible sources of infection—ear, scalp, mouth, tonsil, etc.—are examined.

Other factors being equal (a) The complete absence of cervical adenitis is in favour of the swelling being a branchial cyst, (b) It is unlikely, but not impossible that a tuberculous abscess would exist for many months without the skin becoming involved.

ii *From Cystic Hygroma (cavernous lymphangioma)*—Branchial cyst is opaque with very few exceptions. Cystic hygroma is translucent. This sign alone is sufficient to render differential diagnosis possible. Cystic hygroma is most commonly situated in the lower half of the neck. It is usually diffuse, loculated and first noticed in infancy.

iii *From Solitary Lymph Cyst*—Solitary lymph cyst is invariably translucent. It is most commonly situated in the supraclavicular triangle. This condition is probably allied to cystic hygroma. It usually however makes its appearance in adult life.

iv *From a Deep-seated Venous Hemangioma*—This may prove a veritable trip. Differential diagnosis is rendered possible by employing the sign of emptying. If the cystic swelling under consideration be a venous hemangioma then pressure will cause it to decrease in size and when the pressure is removed it slowly re-fills. It must be borne in mind that it is possible for a large branchial cyst with deep ramifications to give this sign by emptying into the retropharyngeal space, therefore while exercising this pressure the pharynx should be watched before pronouncing the sign of emptying positive.

* It is interesting to compare the position of the cyst with that of congenital branchial fistula. The orifice of the fistula (in the five cases I have been able to muster) was situated in the lower third of the neck—opposite the anterior border of the sternomastoid the tract led upward. In one case the fistula was bilateral.

x *From Chronic Retropharyngeal Abscess*—Coakley¹⁰ (1904) was the first to recognize this possibility. His case was one of retropharyngeal swelling which had been opened three times and 'pus' evacuated. On a diagnosis of tuberculous retropharyngeal abscess, he began to operate. Opening the swelling from the pharynx a large quantity of pus-like fluid was evacuated. Introducing a finger into the cavity he was astonished to find it passed upwards to the atlas and outwards to the great vessels. A piece of smooth-lined wall was taken for section. Pathological report was "Squamous epithelium on a basis of lymphoid tissue."

xi *From Lipoma*—In all parts of the body the diagnosis of lipoma, in its usual situation viz, superficial to the fascia, is an elementary problem. The diagnosis of sub-fascial lipoma, on the other hand, is notoriously difficult. If the differential diagnosis lies between a subfascial lipoma and branchial cyst, it can be immediately settled by the insertion of a needle, for branchial fluid is hardly ever too thick for aspiration.

xii *From Cystic Degeneration of a Malignant Neoplasm*—A rapidly-growing primary or secondary malignant neoplasm in the 'branchial position', undergoing cystic degeneration, sometimes enters the question. It is, however, very unusual for a branchial cyst to make its first appearance at the carcinomatous age.

xiii *From Thyroglossal Cyst*—Thyroglossal cyst need not necessarily be in the middle line. The levator glandulae thyroideae in dissecting-room subjects is most often to the left of the middle line, a portion of the thyroglossal tract therefore is frequently on the left ala of the thyroid cartilage. Thyroglossal cyst usually bears no relationship to the sternomastoid. It never proceeds from the deep surface of that muscle. Microscopically, branchial and thyroglossal cysts are often impossible to distinguish.

xiv *From Myxomatous Degeneration of a Mixed Parotid Tumour*—This is a pathological curiosity. Two cases, diagnosed respectively as adenoma of parotid and cystic degeneration of mixed tumour, proved on section to be branchial cysts (evidently from the first cleft). An intraparotid branchial cyst has been described by Fredet.¹¹

xv *From Anemysm*—This is a theoretical question only, but may be included for the sake of completeness. There is no record of a case of branchial cyst which was pulsatile from transmitted impulse of the carotids.

Having excluded these possibilities the mental process is somewhat as follows—

Here is a patient, age 23, who for eighteen months has had a painless non-translucent cystic swelling which cannot be made to 'empty'. The overlying skin is quite normal. There are no enlarged cervical glands in any of the triangles of the neck. The swelling lies beneath the upper half of the sternomastoid, protruding around its anterior border, beneath the cervical fascia. The patient otherwise is healthy. Then it is *highly probable* this is a case of branchial cyst.

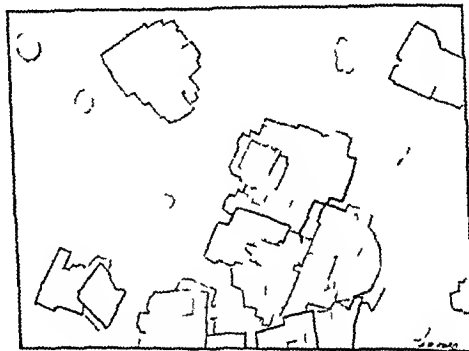


FIG 496.—Specimen of branchial fluid showing cholesterol and epithelial cells.

5 **A Scientific Confirmatory Test**—Introduce an aspirating syringe and remove some of the fluid. Send some to the bacteriological laboratory if thought fit, but place some on a slide and look at a fresh unstained film (Fig 496).

All the cases examined by this method have given a typical picture, viz, cholesterol and squamous epithelium cells. In only 1 of 8 cases examined was cholesterol absent. This could easily be explained. The patient (previously treated as a case of tuberculous abscess) had had the cyst aspirated and injected with an emulsion containing glycerol, which dissolved the cholesterol crystals. Even in this case there were abundant flattened epithelium cells.

7 **An Exploratory Operation**—It has been written in a modern text-book¹² that the diagnosis of branchial cyst can seldom be made before operation. This paper is an endeavour to show that by the adoption of simple general principles it should be quite exceptional for a surgeon to fail to make a pre-operative diagnosis of branchial cyst.

In conclusion, my thanks are due to my teachers, the surgeons of the London Hospital and Liverpool Royal Infirmary, for permission to make use of their cases.

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INSTRUCTIVE MISTAKE

INJECTION OF ALCOHOL INTO THE GASSERIAN GANGLION, FOLLOWED BY WIDESPREAD CRANIAL NERVE PARALYSIS AND THE LOSS OF AN EYE

X Y, age 42, had suffered from trigeminal neuralgia on the right side since 1917. He had had some eight to ten injections of alcohol previous to the one which is the subject of the present note. Each injection had been followed by about three months' relief of pain. He was anxious to have an injection which would give more lasting benefit. On Dec. 22, 1921, he was given general anaesthesia (as on former occasions), and Schlosser's intrabuccal route of injection was used. The needle was felt to slip into the foramen ovale, and was pushed on for about 0.5 cm. A little clear fluid escaped from the needle, evidently cerebrospinal fluid, the needle was therefore slightly withdrawn and 1 cc. of 90 per cent alcohol was slowly injected. The patient was sent back to bed, where he lay on his left side. The next day it was evident that he was suffering from almost complete paralysis of all the cranial nerves except the second pair. He had lost smell, taste, and hearing, he had complete ophthalmoplegia and double facial paralysis, and the movements of swallowing and articulation were impaired. Within one week it appeared that the nerves on the left side of the head were more profoundly affected than those on the right. On the right side facial and eye movements soon returned and he could hear a little in the right ear. The left eye became the subject of ulcerative keratitis and in spite of the utmost care the cornea perforated and the eye had to be removed.

The present condition of the cranial nerves fifteen months after the operation, is as follows —

- I — Loss of smell
- II — Vision in right eye normal
- III, IV, VI — Normal on right side
- V — Complete anaesthesia of face and scalp as far as the vertex. Paralysis of left jaw muscles
- VII — Normal on right. Paralysed on left
- VIII — Right ear. Can hear sharp musical sounds—e.g. a bicycle bell—but no voice sounds. Left ear. Complete deafness
- IX — Loss of taste
- X, XI, XII — Normal function

It is evident that the alcohol must have penetrated into the subarachnoid space at the base of the brain. The greater degree in which the left nerves suffered was due to the fact that the patient lay on his left side after the injection.

Three practical points are suggested by consideration of this case. First, that alcohol injection of the Gasserian ganglion is fraught with serious danger. Second, that the operation ought not to be done under general anaesthesia. Third, that if the needle after penetrating the foramen ovale draws cerebrospinal fluid, the injection of alcohol ought not to be proceeded with.

SHORT NOTES OF RARE OR OBSCURE CASES

SPONTANEOUS RUPTURE OF A HYDRONEPHROSIS

By W. Q. WOOD, EDINBURGH

THE patient was a spare, unmarried woman of 38. She had been aware of a swelling in the left side of the abdomen for the previous ten years, which she had been informed was an enlarged spleen. She suffered some inconvenience from a constant sense of weight in the region of the swelling and occasionally from aching pain in the left side, but in spite of this she was able to lead an active life, though she always had a desire to sit down, on account of the feeling of weight in the abdomen. Apart from occasional nocturnal frequency of micturition, she had never had any renal or bladder symptoms, and had never noticed anything abnormal about the urine.

On the night of Jan. 16, 1922, instead of going to bed in her usual sedate fashion, she jumped into bed and was immediately seized with agonizing pain in the abdomen, which was followed by vomiting. She was seen shortly afterwards by her medical attendant, and promptly sent to Chalmers' Hospital. She was seen there about an hour and a half after the onset of symptoms. She was then in the most acute distress, continually calling out and writhing about on account of the severity of the pain. She could not endure an abdominal examination, and it was impossible to make out more than a general abdominal tenderness and rigidity. The temperature was subnormal (97°) and the pulse rapid. Under general anaesthesia, it was evident that the abdomen was moderately distended. This distention appeared to be general, and no definite localized swelling could be made out. It was thought that the condition might possibly be a ruptured ovarian cyst.

OPERATION.—A mid-line incision was made below the umbilicus, and on opening the abdomen a large retroperitoneal swelling was found. It extended from the diaphragm above to the pelvic brim below, and was mainly on the left side. The retroperitoneal tissue, visible through the peritoneum, presented a curious oedematous appearance. A needle was introduced to ascertain, if possible, the nature of the swelling, but no fluid could be drawn off. The peritoneum was then incised on the lateral side of the descending colon and when the colon was turned medially and the water-logged extraperitoneal tissue brushed aside, a cyst wall was discovered behind. This turned out to be the wall of a large hydronephrotic sac with a rupture towards the lateral side (Fig. 497). The sac was readily separated from its surroundings and removed, after the pedicle formed by the renal vessels had been secured. A tube was brought out in the loin from the space which the cyst had occupied, the parietal peritoneum repaired, and the abdomen closed.

The patient made an uneventful recovery and left hospital on the twenty-third day after operation. When seen recently (Nov. 19, 1922) she expressed herself as feeling perfectly well. She has had no urinary symptoms since the operation and feels much fitter since being relieved of the abdominal swelling.

The hydronephrotic sac before rupture was of a large size—probably about that of an adult human cranium. The renal tissue appeared to be entirely destroyed except towards the upper end where the wall of the sac was a little thicker than elsewhere. The outlines of the original calices could still be made out in the form of localized sacculations, but most of the specimen consisted of a smooth thin walled cyst.

This case appears to be a rare termination of a hydronephrosis. The exciting cause of the rupture must have been the sudden increase of intra-abdominal tension when the patient jumped into bed, so that the rupture might perhaps, be called spontaneous. Rupture from an actual trauma occasionally occurs. Nine cases were recorded by Oehme,¹ in 1907, and several have been noted since. Of Oehme's cases, 8 were operated upon. Of 3 transperitoneal nephrectomies, 2 died, of 4 lumbar, 1 died. One nephrectomy by the combined method was followed by death.



Fig. 10.—The hydronephrotic sac has been laid open from the front. The remains of the calices can be made out in the interior. The large rent (R) is seen towards the lateral border.
 Reproduced from a drawing by Mrs. H. Q. Wood.

In the present case the favourable result is probably to be attributed to the absence of other injury and to the promptitude of the medical attendant in sending the patient to hospital, the patient being operated on about two hours after rupture. The only difficulty in the course of the operation arose from venous hemorrhage, which occurred while the sac was being stripped from its surroundings. To check this, a large moist towel was packed into the lumbar region and the pedicle secured as quickly as possible.

It is interesting to note that the remaining kidney appears to be functioning perfectly.

REFERENCE

¹Oehme: Beitr. z. Klin. Chir., 1907, li, 715.

AN OBSCURE CERVICAL GROWTH

By WALTER MERCER EDINBURGH

HISTORY—R F A youth, age 20, whom I saw some months ago in consultation. While on service in India on Sept 29, 1920, the patient was taking part in a tug-of-war competition, when the rope was let go by one side and the loose end recoiled, giving him a severe blow on the right side of the neck. Beyond bruising, and pain on movement, he was comparatively well for a few days, when the pain became very much worse and he had to be admitted to hospital.

ON EXAMINATION—Oct 20, 1920. Patient was lying flat on his back and unable to move, apparently for fear of exciting pain. The temperature was raised and swinging and the pulse-rate increased. He had to have everything done for him including his feeding. All movements caused pain, particularly those of his neck and right arm, where



FIG 198—Showing the extent of bony outgrowth in the vicinity of the 5th, 6th and 7th cervical vertebrae.

it was excruciating, and caused him to shout loudly. His neck was swollen and tender on the right side, and it was impossible to elicit any movement in it. The right arm was swollen and tender, and especially so along the lines of the nerves. There was severe neuralgic pain in these nerves, constant in character. The skin of the lower arm was tender to the touch. The right arm and face as far as the middle line were often pale in colour and covered with perspiration, while the rest of the body was quite dry. The right pupil was dilated. The lower limbs were normal, although their movements were slow and done with difficulty, and he had occasional referred pain in them. The abdominal reflexes were normal, and he had control of his urine and feces.

Nov 15. X rays showed a tumour of cancellous bone attached to the right side of the 6th cervical vertebra about the size of a Tangerine orange (Fig 198).

LATER HISTORY—During the next few months the signs and symptoms progressively increased, the pain continuing severe, and the forearm and hand beginning to show signs of loss of trophic influence, the skin being glazed and the nails cracking. His pulse and temperature were little affected. Blood-count showed a polymorphonuclear leucocytosis of 12,000. He lost weight rapidly, and the continuous pain necessitated the repeated use of morphia. He was invalided home to this country with the diagnosis of tuberculous spinal caries of the 6th cervical vertebra.

As the signs and symptoms were increasing in severity and the tumour was growing in size, the case was considered later to be a sarcomatous tumour.

Up to this time little had been done in the way of treatment beyond that for the pain, but now a rigid neck support was fitted to him, and from this time onwards the symptoms improved. The pain lessened, and he was able to be got into a wheeled carriage and into the open air. In a few months the swelling in the neck became less. The blood-count improved, and the morphia was gradually stopped. The arm condition became normal, although there remained some stiffness of the joints from long-continued disuse. He was then encouraged to stand, and gradually to take a few steps.

PRESENT CONDITION—The patient now has no pain, but there is still a swelling in the neck on the right side. This is tender to pressure. The neuritis has completely disappeared, but there remains some slight stiffness in the finger-joints and weakness in the whole right arm. The most recent skiagram—June, 1922—is reproduced, and shows the extent of the bony outgrowth in the vicinity of the 5th, 6th, and 7th cervical vertebrae. It is interesting to see that it has extended to the left side further up in the cervical vertebrae.

CONCLUSIONS

It would seem reasonable to infer that a periostitis was set up by the injury from the rope, and that this was infected with organisms of a low virulence. The only treatment that benefited the condition was the immobilization of the neck. As soon as this was done the patient improved, and morphia was gradually diminished, his immunity increased, and he got the better of the inflammatory condition.

I have to thank Major Maurice Sinclair for permission to publish his notes of the history of the case.

INTERNAL HERNIA FOLLOWING POSTERIOR GASTRO-ENTEROSTOMY WITH ACUTE DILATATION OF THE STOMACH AS A SEQUENCE TO REDUCTION

By W. TURNER WARWICK, LONDON

ALTHOUGH the occurrence of internal hernia as a sequence of gastro-enterostomy has been recognized as a possibility almost ever since the operation was first performed, the condition is nevertheless so rare that the following case, taken from the records of the Middlesex Hospital, is of interest.

A T, age 59, ex-policeman, was admitted to a medical ward of the Middlesex Hospital in April, 1917, complaining of chronic abdominal pain. For the previous two or three years the patient had had pain in the epigastric region, which was relieved by taking food and by vomiting. Sickness occurred at any time, with no special relation to meals. Hæmitemesis was an occasional symptom, but was not marked. The bowels were regular. The patient was a well-built man, but somewhat thin and anæmic. The appearance of the abdomen was described as scaphoid, but on clinical examination no abnormal signs were noted. A diagnosis of duodenal ulcer was made, and the patient was transferred to a surgical ward. A laparotomy confirmed the diagnosis of duodenal ulcer, and a posterior gastro-enterostomy was done. The edges of the opening in the transverse

mesocolon were not sutured to the stomach or jejunum. The patient made an uninterrupted recovery, and left the hospital three weeks later.

In May, 1919, the patient returned to hospital, stating that three months previously he had had an acute attack of pain in the pit of the stomach which lasted some days, and that since then he had a continual dull ache on the right side of the abdomen, and across the small of his back. The pain which he had experienced previous to his operation had never recurred, and there had been no further hæmatemesis. He now suffered from flatulence, but was never sick. Since the operation he had noticed that his abdomen had begun to swell, and constipation had developed. On examination the abdomen was now found to be very protuberant, but as the patient had gained in weight since the operation, this was thought to be due to fat. There was no visible peristalsis, nor was there any fluid in the abdominal cavity. The scar was linear and well healed. The constipation was relieved by treatment, but the pain had not entirely disappeared on his discharge three weeks later, and was thought to be functional.

In 1920 he was admitted again to the surgical wards, still complaining of pain which he now described as dragging, in the middle of the abdomen. Since leaving hospital in 1919 he had been for some weeks an in-patient in another London hospital, but his condition had not been permanently relieved.

On examination, the abdomen was distended, but showed no rigidity or tenderness on palpation. The physical signs revealed nothing definite, but in view of the persisting symptoms an operation was decided on.

OPERATION—On opening the abdomen, it was found that the whole of the small intestine with the exception of the terminal 18 inches of the ileum had herniated into the lesser sac through the opening in the mesocolon left at the previous operation. The gut was withdrawn from the sac without difficulty, and the opening closed by suturing the edges to the line of the anastomosis.

For some days after the operation the patient seemed to be progressing favourably, although vomiting of small amounts occurred at frequent intervals. The distention of the abdomen did not seem greater than before the operation. On the ninth day vomiting was more copious, and the general condition became much worse. Death occurred on the following day.

POST-MORTEM EXAMINATION—This revealed an acute dilatation of the stomach. This organ filled the abdominal cavity, and the left arch of the diaphragm was considerably displaced upwards. The lower lobe of the left lung was collapsed, and the mediastinal contents were pushed over to the right. The dilatation was confined to the stomach, no part of the duodenum being affected. No sign of the old ulcer was found, nor was any other abnormal condition present in the abdomen or elsewhere.

Internal hernie occurring after the operation of gastro-enterostomy are of two varieties.

1 The most widely recognized type takes place through an unclosed opening in the transverse mesocolon, as in the above case. In this type the whole of the small intestine may find its way through the aperture.

Moyulhan¹ describes two cases illustrating the clinical sequence of events which may result from such a hernia. In the first case death occurred on the tenth day with symptoms pointing to intestinal obstruction. A similar hernia occurred in a second case, more gradual in onset, and was operated on a year later. In this case the only prominent symptom was persistent vomiting. No mention is made here of abdominal distention. Paterson² states that since the adoption of the practice of suturing the edges of the mesocolon to the stomach or duodenum, no case of this complication has been recorded. A hernia similar to this in type may occur after resection of a portion of intestine, through a gap in the mesentery, when the edges are not carefully sutured. It has also been noted through congenital apertures in the mesentery and mesocolon.³

2 In the second variety of hernia, coils of intestine pass over the loop formed by that portion of the jejunum between the duodenojejunal flexure and the site of the gastro-

enterostomy. The possibility of its occurrence, therefore, would seem greater in the anterior operation than in the posterior modification, where the loop is so much shorter. This variety has only been described in 12 cases,⁴⁻⁶ of which 7 at least followed a posterior gastro enterostomy (5 short-loop, 2 long-loop), 3 an anterior, while in 2 the type of operation is not stated. This preponderance in the posterior modification is doubtless due to the much greater frequency with which this operation is performed.

The possibility of such a hernia also exists after anastomoses between the stomach and jejunum when a partial gastrectomy is done although no cases have been recorded. A hernia similar in type may also occur rarely after a colostomy, round the loop of the pelvic colon, and between it and the adjacent left parietal wall.

The methods of preventing this variety of hernia suggest themselves at once and do not call for detailed description. Thus, the space over the loop is very small in the short-loop posterior gastro enterostomy, and can be easily closed by stitches between the jejunum and the adjacent mesocolon. The so-called 'no-loop' method of performing anterior gastro enterostomy described by Sherren lends itself to the same treatment. When the ordinary anterior gastro-enterostomy is performed the closure is more tedious, but suture of the loop to the mesocolon, to the great omentum, and to the stomach will obviate the risk of hernia. The gap round the pelvic colon in colostomy can be prevented by suturing the lateral surface of the mesentery to the parietal peritoneum.

In the above case, the onset of acute dilatation of the stomach after the reduction of the hernia is of interest. The causes of acute dilatation given by Sherren⁷ are (1) Obstruction of the duodenum by the superior mesenteric artery, which crosses it (Rokitansky, Albrecht). An occasional factor in this is an adhesion of the small gut to the pelvis.⁸ (2) Excessive secretion (Fagge, Henry Morris). (3) Paralysis (Campbell Thompson). (4) Septic intoxication.

Sherren also states that paralysis of the stomach is the condition regarded by most modern writers as the primary cause. In the above case the dilatation was confined to the stomach. There was no evidence at the post-mortem examination of compression of the duodenum by the superior mesenteric artery, and no sepsis was present. The explanation which seems most satisfactory is that provided by the paralytic theory. The removal of the support afforded by the crowding of the small intestine in the lesser sac may have played some part in the initiation of the condition.

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A CASE OF BILOCULAR GALL-BLADDER

By BENJAMIN W. RYCROFT, BRADFORD

The specimen which is the subject of this paper was taken from the following case —

Mrs. L., a stout florid woman, age 42, had been troubled for many years by repeated attacks of biliary colic, the first occurring in 1909, the last in May, 1922. She suffered very little discomfort during the intervals, and up to September, 1919 her health had been quite good. In the early part of October, however, whilst chasing a pig, she was seized with a severe constricting pain in the right hypochondrium at the costal margin, which radiated to the epigastrium and round to the right flank. Jaundice supervened, and the patient was confined to bed for six weeks, during which time the pain recurred at intervals

In the following May a severe attack was again experienced, with aggravation of all the symptoms. In November, operation was recommended.

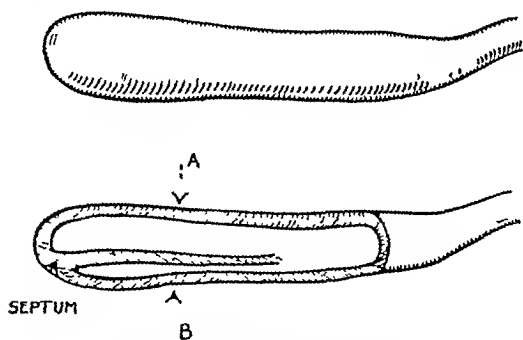


FIG. 499.—Diagrams illustrating the gall bladder. A B shows the plane of section depicted in Fig. 500.

In November, under general anaesthesia of chloroform and ether, cholecystectomy and choledochotomy were performed. Twelve calculi were removed from the gall-bladder, and eight from the common bile-duct. The recovery of the patient was uneventful.

Examination of the Gall-bladder (Figs 499, 500).—

Macroscopic Appearances.—The specimen was tubular in shape, and contracted and firm in consistency, it had very much the appearance of an appendix vermiciformis. On transverse section a septum measuring about $6\frac{1}{2}$ cm. in length and $\frac{1}{2}$ cm. in thickness was found, commencing in the fundus of the gall-bladder and extending along its longitudinal axis. The gall-bladder was thereby divided into two unequal loculi, the diameter of the larger being 8 mm., and of the smaller about 5 mm. A small calculus was found in the smaller loculus.

Microscopic Appearances.—Sections were cut at different levels. Each loculus was lined by columnar cells having basal nuclei. Many tubo-racemose glands were present, and those near the fundus possessed a slight degree of dilatation.

The septum intervening between the loculi was mainly composed of fibromuscular tissue, with glands dotted here and there. At its greatest thickness near the fundus it measured $\frac{1}{2}$ cm.

My best thanks are due to Dr Macnaughton, of Leicester, for permission to publish the case.

At that time there was a yellowish green discoloration of the conjunctivae, skin, and mucous membranes. The faeces were bulky, and putty-like in colour, the urine contained bile pigments in excess, and a trace of bile salts. Physical examination elicited deep tenderness in the right hypochondrium and epigastrium, but there was no hyperaesthesia of the skin. The recti muscles on both sides were rigid in their upper thirds. In October, 1919, the pulse rate was 82, in November 1922, it was 52 per minute. Constipation and pruritus of the skin had been marked in the later stages.

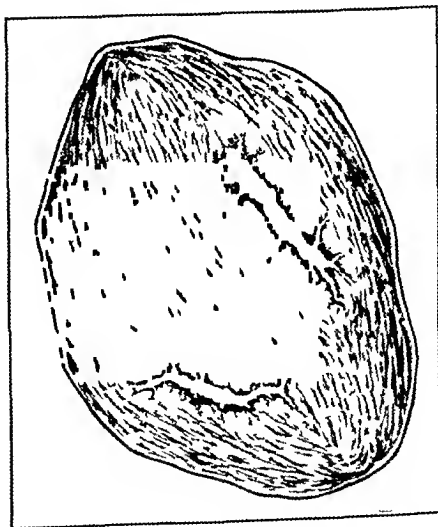


FIG. 500.—Transverse section of gall bladder (x 4).

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FIBROMA OF THE STOMACH

By A. J. BLAXLAND, NORWICH

FIBROMATA are mentioned in certain text-books¹ as occurring in the stomach, but I can find no evidence of the report of a case. Nineteen cases of myoma or fibromyoma of the stomach, however, have been recorded: Some of these were of great size—one weighing 5½ kilograms, and another being the size of a man's head. The marked microscopic resemblance between a fibroma and a fibromyoma makes it reasonable to suppose that some of these recorded cases may really have been pure fibromata.

I have recently had under my care a man from whose stomach I removed a simple solid tumour, which on microscopical examination proved to be a simple fibroma. The interesting point about the case is that, in spite of the weight and size of the tumour (which exceeded that of a billiard ball), there were no symptoms except recent melæna—no discomfort, otherwise, of any description.

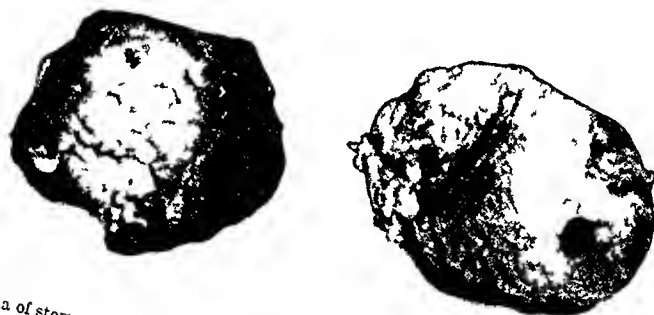


FIG. 501.—Fibroma of stomach. *a* Peritoneal aspect. *b* Showing pits on the mucous surface ($\times 4$).

The patient, a medical man, age 58, moderately stout, and healthy looking, had a fairly severe melæna at the end of September, 1922. A year previously he had had a severe melæna lasting several days, causing marked temporary anæmia. When 16 years old he passed tarry motions for a week, after riding a horse. He had had no other symptoms of any sort—no indigestion, no vomiting.

EXAMINATION.—Four weeks after the last attack of melæna, the patient was obviously somewhat pale, but showed no other abnormal physical signs. There was no tenderness in the abdomen, and no swelling could be felt.

A blood-count showed a mild secondary anæmia. Red cells 4,700,000. Hæmoglobin 70 per cent. Leucocytes 7,000. Radiography after a bismuth meal showed no abnormality in the shape or motility of the stomach.

In consultation with Dr. Burton-Fanning, a provisional diagnosis of duodenal ulcer was made and an exploratory laparotomy was found in the posterior wall of the stomach.

OPERATION.—On Oct. 30, a round tumour was found in the posterior wall of the stomach, its upper border being situated at the centre of the lesser curvature. An opening was made into the lesser sac, just below the greater curvature. The stomach was turned up exposing its posterior surface. There were no adhesions. Clamps were applied on either side of the tumour and the stomach was opened at the lower border of the tumour which was removed by incising round it on either side up to the lesser curvature. The gap thus caused was closed by a double layer of sutures without producing any obvious deformity of the stomach. An uneventful recovery ensued.

DESCRIPTION OF THE SPECIMEN (Fig. 501).—In shape the tumour is almost spherical, its diameter measuring 2½ inches. It is slightly lobular. It is solid but soft, giving an

impression on palpation of being cystic. Its posterior surface is covered by peritoneum, which appears to be very thin in places. The anterior surface is covered with mucous membrane, and presents four moderately deep pits (from which the hemorrhage had presumably arisen). The tumour has a well defined edge, and it projects equally towards the peritoneal and mucous surfaces.

MICROSCOPICAL REPORT FROM DR CLARIDGE, NORWICH.—On examining a microscopic section of the tumour it is found to be richly cellular, composed of spindle shaped elements arranged in interlacing bundles. The general appearance is that seen in a uterine fibroid, but muscle fibres do not appear to be present as they do not stain characteristically by Van Gieson's method. The capsule is well marked and shows no infiltration, so that I think the tumour must be called a fibroma.

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REVIEWS AND NOTICES OF BOOKS

Surgery of the War Edited by MAJOR-GENERAL SIR W. G. MACPHERSON, MAJOR-GENERAL SIR A. A. BOWLEY, MAJOR GENERAL SIR CUTHBERT WALLACE, and COLONEL SIR CRISP ENGLISH. In two volumes. Pp. 618 and 604, with 16 and 7 coloured plates and numerous other illustrations. London: H.M. Stationery Office. 26s. each volume, post free.

This work has been prepared by the consulting surgeons and by some of the surgical specialists who held commissions in the Royal Army Medical Corps during the great war. Its timely appearance within a few years of the termination of the conflict has ensured that the subject of which it treats shall be described when its events are still fresh in our memory.

Volume I—The first volume deals with general subjects affecting the surgery of the war, and indicates the development of front line surgery and wound treatment generally, followed by chapters on wounds of the thorax and the abdomen. The first two chapters describe the various projectiles used by the Allies and their enemies and discuss in general terms the results of projectile action. They are written by Colonel Pileher and shock in front line areas and in casualty clearing stations. A very good summary of the various and conflicting theories as to the nature of shock is given. In regard to treatment, drugs are considered of no value, and in addition to the ordinary methods of warmth, administration of fluids, and sleep, the greatest importance is attached to transfusion, and it is stated that the use of gum aether solution has proved of enormous practical value. Major Gordon Taylor follows with a description of the methods and indications for blood transfusion, indicating the valuable results obtained by these methods both in the treatment of wound shock and after operation.

Gas gangrene is fully described by General Sir Cuthbert Wallace, and the article is illustrated by beautiful coloured plates by Maxwell. Sir Frederick Andrewes writes a short summary of the subject of tetanus, emphasizing the great value of the prophylactic administration of antitoxin, which reduced the incidence of the disease from over 8 per thousand to 0.2 per thousand between September, 1914, and January 1915.

One of the most fascinating sections of the book is comprised in the four chapters which deal with surgical work in field ambulances, the development of casualty clearing stations and front line surgery in France, wound treatment in general hospitals in France and wound treatment in hospitals in the United Kingdom, by Colonel Max Page, General Sir Anthony Bowlby, General Sir George Maxims and Colonel Bond respectively. In these chapters we have vivid pictures of all the great difficulties and how by organization on one hand and research on the other these difficulties were met and overcome. The story of how the treatment of grossly infected wounds passed through many stages, both antiseptic and aseptic, is one which will remain a classic for all time.

The section on wounds of the chest and lungs is by Colonel Gask. It gives with great wealth of illustration, together with operative and mortality statistics, the history of the progress of our knowledge in this subject gained during the war. The necessity for early complete excision of infected wounds with removal of all devitalized tissues and foreign bodies, followed by early closure of the wound, is emphasized. The chapter on injuries to the pericardium and heart is by General Sir George Maxims, and it contains a detailed account of all varieties of gunshot injury noted during the war, illustrated profusely by Maxwell's drawings. The article is accompanied by 39 case reports from various sources of operations, both early and late, done for cardiac wounds.

The concluding chapters of the first volume, dealing with abdominal wounds, Sir Cuthbert Wallace, and present a most complete account of this subject, both from the anatomical and clinical aspects. These articles are of particular value for the many tables and diagrams representing the incidence and results both of gunshot wounds and their treatment. These chapters are so full of valuable information that it is impossible to do justice to them in the space at our disposal. Expectant treatment of gunshot wounds of the abdomen has been definitely superseded by operative treatment, but the mortality still remains very high, being about 50 per cent excluding moribund cases, and about 60 per cent including these, and of all those which made an immediate recovery about 10 per cent died subsequently, whilst only 10 per cent were fit to return to their military duties.

Volume II—The section on wounds of the head is by Captain Trotter and Captain Wiggstaff, and embodies the generally accepted teaching in both early and late conditions. In regard to the

latter, some stress is laid upon unresolved cerebral contusion as a cause of persistent headache, and it is stated that a decompression will cure this. Another point which is perhaps open to difference of opinion refers to the necessity for closing apertures in the skull. The methods advised for this procedure are the use of celluloid plates or a piece of the tibia, whilst no mention is made of the great advantages of the cartilage graft.

The section on the face and jaw is by Major Gilhes, and although it is full of useful diagrams and directions, it is disappointing, because no one injury is dealt with fully enough to give sufficient guide for actual treatment.

The sections on the spine and spinal cord are by the late Sir William Thornburn, and contain a well-balanced summary of both English and Continental views about gunshot injuries to the spine, and especially about the most important subject of the indications for operative treatment. The same author deals with the peripheral nerves in a comparatively short chapter, which makes no attempt to include the lesions of individual nerves.

The next section constitutes one of the most valuable parts of the whole work. It is concerned with injuries to the blood vessels, and is by General Sir George Makins. It is based upon the observation of nearly 1200 cases, the notes of which were specially collected by the author. The pathological details and illustrations, and the great wealth of the clinical reports, will make this article the standard work of reference for some time to come.

The next 200 pages deal with the injuries of bones and joints, together with the general problems of so-called orthopaedic treatment. Colonel Frank in treats of wounds of the joints and fractures of the upper extremity, and Colonel Webb Johnson describes the fractures of the lower extremity. In all these articles full justice is done to the merits and advantages of the Thomas splint. The other orthopaedic articles are written by General Sir Robert Jones and some of those associated with him in this work during the war. Jones himself takes the subject of stiff joints, and his advice as to treatment is on conservative lines. For example, in speaking of bony ankylosis of the knee joint he states that "arthroplasty of the knee has no place in war surgery." This would indicate that the distinguished author does not believe in the results claimed by such workers as Putti. Major Elmslie contributes a useful article on amputations and artificial limbs.

The work concludes with chapters on injuries to the eye and ear by Colonel Lister and Major Scott. Each volume contains an index.

The editors and contributors are to be congratulated on having produced a very useful and authoritative work. The illustrations are of special value. The use of two different qualities of paper is unusual and displeasing, and the numbering of the illustrations separately in each article does not facilitate ready reference.

The Early Diagnosis of the Acute Abdomen. By ZACHARY COPE, M.D., M.S., Surgeon to Out-patients, St Mary's Hospital, etc. Pp 222, with 28 illustrations. 1921. London: Henry Frowde and Hodder and Stoughton. 12s 6d net.

THE fact that the author emphasizes repeatedly throughout the book the extreme importance of recognizing the early signs of the acute abdomen renders the work one of considerable value. Many abdominal diseases, both acute and chronic, are only recognized even to-day by what should be more accurately described as complications rather than signs and symptoms. Distention of the abdomen in peritonitis, and fecal vomiting in intestinal obstruction, are still held by some to be signs of these diseases. They are in reality complications which should never be seen, and, if the author's advice be followed, will be more rarely met with in the future than they have been in the past. No treatment beyond operation by a competent surgeon at the earliest possible moment is discussed, and no details of operations are given. There are descriptions of the signs and symptoms and methods of diagnosis of acute abdominal catastrophes.

Several little known signs, such as the obturator test—internal rotation of the thigh causing pain in cases of pelvic appendicitis with abscess—shoulder pain in inflammatory conditions about the diaphragm, are illustrated and explained whilst hyperæsthesia, diminished liver dullness, and testicular pain are mentioned and their anatomical explanations given.

There are nineteen chapters in the book, the first of which is devoted to the principles of diagnosis. The all important point of early diagnosis and the danger of delay in treatment are plainly indicated. Methods of diagnosis—the history and examination of the patient are treated very fully—illustrations of the psora test, and the method of testing for hyperæsthesia are given.

The chapter on appendicitis is lucidly written, and many points of great importance are emphasized. Murphy's sequence of symptoms is given, and unless this occurs the practitioner should question the accuracy of his diagnosis. The absence of abdominal rigidity and even rise of temperature in pelvic abscess, and the continued acceleration of the pulse as an indication of the onset of peritonitis, are points of importance to which attention cannot too often be drawn. In the differential diagnosis of appendicitis the possibility of the presence of a stone in the right ureter should not be overlooked.

Many points in the chapter on perforated gastric and duodenal ulcers might, we think, be expressed differently. The signs and symptoms are divided into three stages: (1) Primary shock,

(2) Reaction, (3) Frank peritonitis with toxic shock. With the first of these stages we are in entire disagreement the clinical signs and symptoms of shock are well known, and two of the most important are rapid pulse and lowered blood pressure. We venture to say that neither of these signs is present in the early stages of perforated gastric or duodenal ulcer. In several recent cases seen by us, and according to the teaching of Moynihan which is now generally accepted, the pulse has been normal and the blood pressure not lowered. These cases were seen within a quarter of an hour of the perforation. If the author means that there is a rapid pulse and lowered blood pressure (clinical shock) in the first minute after the perforation, we cannot dispute this, as we have not seen cases so soon after the catastrophe. We believe that there is no stage in the perforation of a gastric or duodenal ulcer that shows the clinical signs of shock, and for this reason cannot agree that the three stages into which the author divides the signs and symptoms really exist.

In an earlier part of the work the author says that many if not most patients with a serious acute lesion of the abdomen have a normal pulse for a considerable time during the early stage. This we believe is the correct statement and one which requires the utmost emphasis. Practitioners are inclined to say even to say that a patient cannot have a perforation because the pulse is quite normal. We would strongly endorse the author's statement above, but equally strongly condemn the description of a primary stage of shock in perforation of a gastric or duodenal ulcer. He states that this stage may last an hour or two. With this we do not agree. We have never seen a patient in which there were the clinical signs of shock within an hour of the perforation. We maintain that there are no clinical signs of shock in the early stages of perforation, the pulse and blood pressure are normal. This must be clearly understood, or the time for operation at which the greatest amount of good can be done will be missed.

In the chapter on acute intestinal obstruction, under the heading of diagnosis of small-gut obstruction, the author states among other signs that fecal vomiting may occur with a free, flat, and non tender abdomen. We think that fecal vomiting to any marked extent never takes place unless there is some distention of the abdomen. If intestinal obstruction be left until fecal vomiting appears, the time when the success of an operation may be interpreted with some degree of certainty has already passed.

The chapter on strangulated and obstructed hernia is good, but we do not agree that it is often very difficult to differentiate between strangulated and obstructed hernia. If the hernia is tense it is strangulated, and the author rightly condemns the use of fomentations and ice-bags, and his opinion on this will be endorsed by all who are accustomed to deal with this condition. Strangulated inguinal, femoral, and umbilical hernias are dealt with, and the differential diagnosis of each is given.

There is a chapter on ruptured ectopic gestation, and the signs and symptoms are divided into three. (1) Signs before rupture, (2) Signs at the time of rupture, and (3) Signs after the rupture has taken place. That ectopic gestation should be diagnosed before the rupture just as appendicitis is, is the aim of the author, and with this all practitioners will agree.

Chapter XIII is devoted to cholecystitis and other causes of pain in the right upper quadrant of the abdomen. Under the heading of jaundice the author states that it is not usual in simple cholecystitis, and is not the rule even when gall-stones are present. We would say that a faint tinge of jaundice, often confined to the sclerotics, is by no means infrequent in acute cholecystitis, though it is often so faint that it will be missed unless looked for with care.

In the chapter on abdominal peritonitis we do not think the fact that the symptoms may be long delayed is quite sufficiently emphasized. This is especially the case in intra abdominal rupture of the bladder. In this condition peritonitis may not be set up for many hours, and in quite a number of cases there are very few signs of shock. We do not think the absence of signs and symptoms in the early stages of severe abdominal catastrophe is sufficiently noted, as it is this absence which causes delay in operation or a false feeling of security in the mind of the practitioner. Acute peritonitis the acute abdomen in the tropics diseases which may simulate the acute abdomen acute abdominal disease with genito urinary symptoms, are dealt with in other chapters which convey to the reader exactly what the author intends they should. It is a book from which the student and practitioner can obtain a considerable amount of very valuable information, and one which, if carefully followed, will prevent undue delay in dealing with acute conditions occurring inside the abdomen.

Studien zur Anatomie und Klinik der Prostatahypertrophie. By JULIUS TANDLER and OTTO ZUCKERKANDL. Lange 8vo Pp 130, with 121 illustrations. 1922 Berlin Julius Springer. Price in England 2/3s.

Two authors of this book have worked in collaboration for seventeen years, and have published a long series of papers dealing with various phases of prostatic hypertrophy. The present volume is intended as a resume of their work but is Zuckerkandl's unfortunately died before it was published the survivor has brought it out as a memorial to his dead friend and colleague. After a historical survey of the disease the authors state that, in their opinion, prostatic hypertrophy is due to an idiopathic new growth originating in rudimentary prostatic glands.

situated in the upper portion of the prostatic urethra, i.e., in the portion above the verumontanum. As this growth increases in size, it compresses and flattens out the true prostatic tissue, until this simply forms a capsule round the adenomatous mass. The new growth is firmly united to the urethral mucosa, and is a consequence of this the upper part of the prostatic urethra becomes elongated by the upward development of the tumour, and its antero-posterior diameter is increased in proportion to its increase in size transversely. In fact it soon becomes a scabbard-shaped cleft between the lateral lobes of the tumour mass, the long axis of which forms a well marked angle with the lower portion of the prostatic urethra.

The authors recognize two types of prostatic enlargement, according to the relationship of the upper end of the tumour with the vesical sphincter. In the first, the growth insinuates itself inside the sphincter, and forms a definite projection within the bladder. As this projection increases in size, it dilates the sphincter more and more, but in every case a groove corresponding to the position of the sphincter can be seen in the enucleated specimen. The intravesical projection is only covered by the mucous membrane of the bladder, and may take on various forms, e.g., a spherical tumour overhanging the urethral orifice, or a horse shoe shaped mass surrounding it, but no matter what its size or shape, it always takes its origin from the prostatic urethra. In the second type, the vesical sphincter is not dilated, and the adenomatous mass is entirely sub-vesical. The floor of the bladder is raised upwards, but the relationship of the sphincter to the other elements of the bladder base is not altered. In this type the prostatic adenoma always surrounds the urethra completely.

The effect of back pressure on the urinary organs is next discussed. The changes in the bladder walls, trigone, seminal vesicles, etc., are minutely described, but the most interesting observation recorded is in reference to the ureter. As the prostate enlarges, the distance between the points where the ureter pierces the bladder wall and where the ejaculatory duct enters the prostate is definitely increased. The vis is put on the stretch and pulls on and kinks the ureter at the point where they cross. Many specimens are illustrated in which the ureter is dilated and hypertrophied above this point, but of normal calibre immediately below it. In the clinical portion of the work two cystograms are reproduced, both of which show a distinct kink in the ureters just outside the bladder shadow. A short chapter is devoted to a description of inflammation arising in the enlarged prostate, and another to the development of cancer in the adenomatous nodules.

Up to this point the subject has been treated entirely from the pathological aspect, and the descriptions have been accurate, clear, and exact. There are, however, two criticisms that must occur to every reader. The first is the origin of the adenoma from 'rudimentary prostatic glands'. The existence of such glands is doubtful, and it is difficult to imagine why these glands, if they do exist, remain quiescent for the greater part of the patient's life, and only become pathological in old age. The French writers describe these tumours as arising from the normal urethral, in contradistinction to prostatic, glands, and this explanation appears to be more reasonable. The second criticism is that it is a pity the changes in the kidney due to prostatic obstruction are not described. The only change mentioned is that the renal pelvis and calices are dilated, but from a practical point of view these changes are the most important of all, as the patient's life usually depends on the state of his kidneys.

About a third of the work is devoted to the clinical side of the question. A good description is given of both perineal and suprapubic prostatectomy, and of the various stages of repair after operation. This is followed by a discussion of congenital diverticula of the bladder as a complication of prostatic obstruction. A case of recurrence after prostatectomy is described, which the authors consider to be due to a fresh development of adenoma in a portion of the urethral mucous membrane left behind at the time of operation, but which, from an examination of the illustrations, appears to have had its origin from a small outlying adenoma that had become detached during enucleation. The last chapter is devoted to the question of diagnosis, but is directed chiefly to the examination of the size, shape, and cystoscopic appearances of the enlarged prostate, the appearance of the bladder, and the interpretation of cystograms, etc., while both the clinical symptoms, and the examination of the renal function, are only briefly considered.

In conclusion, the illustrations are the best feature of this work. They are extraordinarily good and clear, and render the book well worth having. The pathological portion is a clear, detailed, and comprehensive description of the changes which take place in the urethra, bladder, ureters, etc., in cases of prostatic hypertrophy. The clinical portion is not nearly so good or useful, and is marred by many strange omissions. For example there is no mention of the pre- or post-operative treatment, the question of a two-stage prostatectomy is dismissed in five lines, and the symptomatology is very inadequately discussed. There is no bibliography, and no reference is given even when statements of other authors are quoted.

La Radiothérapie Profonde. By ISER SOLOVON, Radiologiste de l'Hôpital Sainte Antoine.
Pp 152, with 40 illustrations. 1923. Paris. Masson et Cie. Paper covers 9 fcs net.

This is an excellent little book, well and clearly written. The subject matter is presented in a way that is concise and easily grasped without being dry. The style is excellent and different methods and points of view are clearly stated without bias. The book should be carefully studied by all beginners, and will be of value also to experienced therapists.

The author points out in the opening sentence of his book that rays, if applied in sufficient intensity, induce destructive changes upon all cellular tissues. He then refers to the work of Scitz and Wintz in regard to the dosage required to induce changes of a favourable kind in the treatment of various conditions. He is not in agreement with the very definite statements made in their work on the action upon cancer and other forms of tumours. As a basis for calculation the doses mentioned may be useful, but like many other workers he cannot accept without reservation the statement that 110 per cent of the erythema dose is the lethal dose for cancer.

He calls attention to the theory put forward by Regaud and Nogier of the specific biologic action of different wave lengths, and quotes the evidence in support of this theory. He contrasts it with the views expressed in Fredrich and Krong's well-known work. These workers maintain that the extremity of the biologic action would appear to be independent of the wave lengths of the incident rays. For the same dose absorbed in the mass of tissue the biologic action is the same for filtered and for unfiltered rays. The writer inclines to the latter view, but says no definite statement can yet be made.

The very important matter of the tube used in deep therapy is entered into, several types are described. The boiling water tube so much used until recently in Germany is discussed. The Liliensfeld tube is very favourably commented upon, there is no doubt that this tube possesses certain advantages over the others.

The physics of radiation therapy has been exhaustively dealt with by the author, the absorption of the rays by the filter, and the value in relation to a standard, such as water, are described. Water is then taken as a standard of comparison with the human tissues, and though the relation is not absolute, it is yet sufficiently near to enable the author to give useful indications for the calculation of the depth dose.

The very important matter of the divergence of the rays in the medium used is dealt with in a very lucid manner. The scattering of the rays in the tissues furnishes a very important factor in deep ray therapy. Valuable tables of absorption values are given. These are entirely useful to the ray therapist.

An important technical point is discussed in a lucid manner. Multiplication of the ports of entry or cross fire has been engaging the attention of workers for years, this is briefly explained. Various methods for determining the coefficient of radio are described and their value indicated. The apparatus used in deep therapy is fully described. Those designed in various centres are detailed, and a full explanation given of the intricate structure of the most popular types. The latest developments of the French technicians are referred to, and their advantages clearly put forth. The apparatus shown on page 47 gives an illustration of a complete installation. This indicates the great developments which have been made in this direction.

The important question of the measurement of the radiations is admirably dealt with, the various methods are outlined, and an excellent description is given of the ionization method. The ionometer designed by the author is fully described in the text, and the description is made easy by the inclusion of a number of good diagrams.

Propedeutique et Technique Urologiques By Dr G. WEINER, Bruxelles. Large 8vo. Pp 492, with 153 illustrations. 1922. Paris: Masson et Cie. 40 fr.

This book is devoted entirely to descriptions of the various methods employed in the investigation of cases of disease affecting the genital and urinary tract. The next chapter deals in detail with cystoscopes, and genital secretions in health and disease. After a description of the urethroscopes, bougies, and other instruments used by the urologist, and the indications for their employment there follow chapters on the examination of each of the individual organs of the genital system. These are the most valuable part of the book, for the many variations met with in disease are well described, and many useful points in technique are given which simplify the investigation of difficult cases and aid in forming a correct diagnosis.

The earlier part of the book is equally well written, but does not contain much that will help the surgeon who specializes in this branch of surgery, and seems hardly likely to be read by others. There are some useful diagrams.

The Thyroid Gland Clinics of GEORGE W. CRILE and ASSOCIATES. Edited by AMY F. ROWLAND. Pp 288 with 106 illustrations. 1922. Philadelphia and London: W. B. Saunders Co. 24s net.

A series of papers on various problems connected with the surgery of the thyroid gland is gathered together in this volume. Each contribution is by an expert in his own line of work, and the collection contains much information of interest to every medical man, though it will be found of particular value to those who operate upon the thyroid gland.

The opening chapters by G W Crile, are of a highly theoretical nature as, for instance, that on the role of the adrenals in exophthalmic goitre, and the ideas contained in them are not always easy to follow. But their author disarms criticism by explaining in the introduction that the volume is an ephemeral work, and only represents the views held in the Crile clinic at the moment of publication. The opinions expressed will be subject to revision, or may even be reversed. Many subsequent chapters, however, are eminently practical and instructive. The pathology of the thyroid gland is well presented, and there is a good account of laryngeal function in relation to the thyroid gland, attention being directed to the frequent occurrence of pre operative abductor paralysis. Intrathoracic goitre, the value of observations on the basal metabolic rate, and the possibility of preventing the development of colloid goitre are all well discussed.

Most readers, perhaps, will find the chief interest of the book to lie in the account that is given of the pre and post operative care of patients with exophthalmic goitre. Great attention is given to the subject of anaesthesia, and the importance of this is not exaggerated. Stress is also laid on the prevention of operative shock, methods now well known in association with the name of Crile being described.

Some account is given of the organization of the work done in the operating theatre. In the Crile clinic this is brought to a state of efficiency such as can only be attained where large numbers of similar cases are constantly being operated upon, though it may surely serve as a useful object lesson wherever surgery is practised. Every surgeon has his own preferences in the details of operative technique, and will probably find something that is not to his mind in the methods used by others, but he cannot fail to be interested in those practised by surgeons so expert as Crile and his associates, beyond this, technical procedures and their results must be seen to be properly appreciated or criticized.

As already mentioned, the claims made for this volume are modest, yet most surgeons will regard it as a valuable addition to their libraries, and those who have enjoyed the friendship of G W Crile will think, as they read his words, that they can almost hear again the genial tones of his voice and feel the influence of his inspiring presence.

The New Physiology in Surgical and General Practice By A RENDLE SHORT, M.D., B.Sc., F.R.C.S. Fifth edition. Crown 8vo, revised and enlarged. Pp 330. 1922. Bristol: John Wright & Sons Ltd. 9s 6d net.

It is difficult to find fault with a book which, in spite of the years of the war, has run to a fifth edition since first published in 1911. Its popularity is equally great among medical practitioners and among students reading for higher degrees in medicine and surgery.

In the present edition three new chapters appear, devoted to the physiology of muscular exercise, the functions of the kidney, and the dietetic factor in the causation of appendicitis. The last named subject depends largely on the work in which the author himself has been especially interested, and hence is more fully dealt with than is perhaps quite justifiable in the present state of our knowledge. Most of the systems of the body are included under the various chapter headings, but although the new work on the heart is adequately reviewed by Dr C E K Herpath, the lungs do not come in for special mention. The newer methods of oxygen administration, to mention one item only, would certainly be of interest to medical practitioners, few of whom have learned to get the full value from such treatment.

The references which appear at the end of every chapter are useful to those who may wish to go further into the subject discussed. After the section on tests for renal function, mention might have been made of Professor Hugh Maclean's little book, which gives a particularly clear and concise account of the subject.

A very few errors have been noticed in perusing the book, an amusing one being the conferring of a title (no doubt well deserved!) on a well known neurologist who has written on aphasia.

The style in which the book is written is really excellent, and the reader's interest never flags. We agree that for medical men who are keenly interested in their work, this book must be as interesting as any novel.

Mistakes and Accidents of Surgery By HAROLD BURROWS, C.B.E., M.B., B.S. (Lond.), F.R.C.S. Demy 8vo. Pp 470 + viii. 1923. London: Baillière, Tindall & Cox. 10s 6d net.

As we know the reputation both of the author and of the publishers, we can readily believe the latter's disclaimer that publicity in the lay press was not sought by either, but the pity is that such a disclaimer should be necessary. As we glanced through the volume—and it was purposely done far more carefully than usual—the thought 'What on earth was this book written for?' constantly recurred to the mind. It is impossible to regard it as a serious scientific work, as claimed by the publishers, for if it is to be judged by this standard, the verdict can only be that it is trivial and cursory. Then again, for whom was it written? Mr Burrows cannot have written this book for his peers, or even for those beginning the practice of surgery in the orthodox way at a large hospital. Is it for the type of would be operator with no surgical training? If so, it

is a very dangerous diet. Many of the so called mistakes and accidents, if they could possibly occur, could only be regarded as crimes, in truth, "Mistakes and Accidents of Surgery" is a misnomer. The book is a conglomeration of physical signs, cases briefly outlined, differential diagnosis—with some mistakes and not a few points to be avoided, much of it is perfectly sound, but it is written as if it were a shorthand report of bedside remarks which had escaped the editor's blue pencil. The surface of many subjects is skimmed and just at the point where one's interest is aroused, another subject is introduced. We are compelled to state with regret that this book is quite unworthy of the reputation of its author.

Lawson Tait his Life and Work A Contribution to the History of Abdominal Surgery and Gynecology. By W. J. STEWART MCKAY, M.B., M.Ch. Royal 8vo Pp 578 + vii, with 34 plates. 1922 London Bailière, Tindall & Cox 25s net

This readable book, which in spite of its size is light and convenient to handle, is written by Dr Stewart McKay, of Sydney, New South Wales, who was formerly one of Lawson Tait's assistants. It is a history of the development of modern gynecology strung upon the thread of a life of Lawson Tait. Dr McKay Lawson Tait was a hero, and he naturally puts forward the best side of his character and work. He shows how much surgery was indebted to a man of comparative little culture, who was a consummate operator and a bold surgeon. In reading his life the thought constantly recurs how much more he might have done, had he been well jostled at a large public school, if he had taken advantage of his opportunities to gain a knowledge of science at the University of Edinburgh, and if he had been attached to a hospital with a medical school where he would have been subject to the daily criticism of his colleagues and the students. The good points in his character—and they were numerous—would have stood him in better stead than they did, he would have saved himself many bitter quarrels, and he would perhaps have lived longer, for he died at the early age of 54, leaving much work to be done. But he lived his own life as a pioneer in gynecology, pointing the way towards the modern surgery of the abdomen, and practising aseptic surgery while hardly knowing more about it than that it gave him good results, and was antagonistic to the methods recommended by Lister. His work was empirical, based to a large extent on trial and error, lacking in a recognition of the broad basic principles which a better education might have enabled him to discover.

Dr McKay has adopted a very comprehensive plan in dealing with his subject. He first examines each important part of Tait's work in detail and then gives the modern views in order to show where the original observations have been confirmed and where experience has led to the adoption of different conclusions. By following this method the history of gynecology is told from the first removal of a diseased ovary in 1872 until the evolution of hysteromyomectomy and the work done between 1891 and 1899. Interspersed with the general history are accounts of the results arrived at by Dr McKay himself as a practising gynecologist in Sydney which render the book additionally valuable. In looking back over the early period of the evolution of gynecology it is deplorable to observe how much time was wasted and how much angry feeling was aroused by simple questions of priority and by the discussions as to the length of an incision time which would have been spent to greater advantage in the elucidation of principles and the study of results rather than of tables of mortality.

Dr McKay concludes with an interesting account of Tait's personal habits and methods of work. He has observed them whilst acting as his assistant in Birmingham from 1891.

Guy's Hospital Reports Vol 71 (Vol 2 Fourth Series) No 1 Edited by ARTHUR F. HURST, M.D. London Henry Frowde and Hodder & Stoughton Subscription £2 2s 0d per annum Single numbers 12s 6d net

GUY'S may be congratulated on their 'Reports', they are good and stimulating. There is one historical article on Astley Cooper by Sir Charters Symonds which contains a good picture of the state of surgery and of medical education a hundred years ago. Osborn contributes a study of case sheets of splenic aneurysm and splenomegaly, together with a good bibliography. Other articles are on "Summer Diarrhoea", "Knock Knees", and "Vicious Circle". Nicholson writes on "The Heteromorphoses in the Human Body" which is intriguing and suggestive of advance in our knowledge of the growth and development of cells.

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